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THE UNIVERSITY OF ALBERTA

THE EFFECTS OF VARIOUS EXERCISE REGIMES ON GROWTH, DEVELOPMENT,
AND MEAT QUALITY IN SHEEP

by

JENNIFER LYNN AALHUS

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH
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OF DOCTOR OF PHILOSOPHY

IN

ANIMAL GROWTH AND DEVELOPMENT

DEPARTMENT OF ANIMAL SCIENCE

EDMONTON, ALBERTA

SPRING 1988

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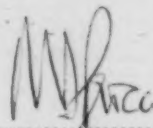
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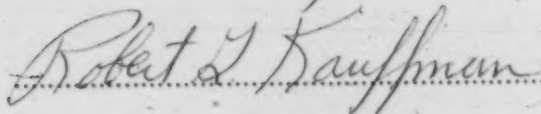
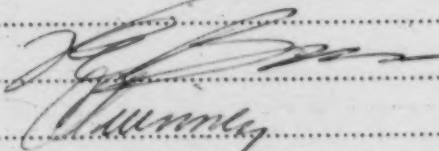
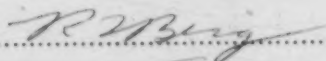
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Supervisor



External Examiner

Date 1988 April 19

DEDICATION

This thesis is dedicated to the pioneer spirit of my parents and grandparents:

Dorothy Dockeray

Sten Aalhus

Esther Holmes

Berta Carlström

John Dockeray

Olaf Aalhus

To adventure, hard work and stubbornness.

ABSTRACT

Studies on the effects of exercise on humans and laboratory animals often fail to consider changes in tissue distribution which occur due to differences in live weight, stage of maturity and individual tissue weights. Meat animals provide an unique opportunity to separate the effects of exercise from other factors which affect tissue growth and development. Four experiments were conducted to investigate the effects of exercise on live animal performance, growth and development of carcass tissues, and meat quality in young, growing, male sheep: (1) static load-carrying, in which sheep carried up to 65% of body weight on restricted feed; (2) a second study of static load-carrying, with *ad libitum* feed; (3) a progressive-resistance exercise, in which sheep were required to jump onto platforms through a circular course; (4) a treadmill endurance exercise.

Low-intensity exercise increased feed consumption but did not influence average daily gains or the proportion of muscle, bone or fat in the carcass. Conversely, high-intensity exercise depressed appetite and reduced average daily gains. The resulting lower live weight at slaughter caused differences in composition normally seen in lighter carcasses; an increased proportion of muscle and bone and a decreased proportion of fat. The effects of exercise on the distribution of muscle and bone were two-fold: (1) a direct growth stimulating effect on individual muscles and bones involved in the exercise; (2) a growth acceleration towards a mature muscle and bone distribution, independent of involvement with the exercise.

Dissectible fat weight and distribution were not affected by exercise; however, chemically determined intramuscular fat decreased. Unexpectedly, the weight of the *ligamentum nuchae* increased with high-intensity exercise and decreased with low-intensity exercise. Exercised sheep had consistently more tender meat; this was associated with a higher myofibrillar protein content and a lower collagen content. The shifts in connective tissue weights at the gross level and the lower collagen content within the muscle indicate that exercise may play an important role in connective tissue metabolism. At the cellular level, muscles from endurance-exercised sheep had a higher proportion of slow-oxidative fibers, shorter sarcomeres and lower rates of post-mortem pH decline. The shift to a more aerobic

metabolism is very similar to changes in fiber distribution which normally occur with advancing maturity.

Although exercise offers few benefits to meat production the increase in tenderness as a result of exercise is important and warrants further investigation. These studies indicate that subjective measurements of the effects of exercise in humans and in laboratory animals should be interpreted with caution.

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Although, at times this study seemed long and tedious, the people that I worked with made it all worthwhile. For their help, good humor and expertise I would like to express my heartfelt appreciation to the following people. At the Metabolic Unit: Jack Francis, Paul Gregory, and Charlane Gorsak. At the Meat Lab: Inez Gordon, Helen Murphy, and Bruce Peterson. At the Feed Mill: Bud Pringle. At the Farm Shop: Shawn Kallal. In the labs: Terry Fenton, Anya Nowinska, Kay Olsen, Ron Pelechaty, Julian Daniels, Brian Turner, Brian Kerrigan, and Sterling Worth. On the computer: Ray Weingardt and Gavin Godby. The summer students who helped: Jim Townshend and Verna Devall.

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Chapter I

EXERCISE: A REVIEW OF ITS EFFECTS ON GROWTH AND DEVELOPMENT FROM A MEAT PRODUCTION PERSPECTIVE

Although there is a wealth of information on the effects of different types of exercise on humans and laboratory animals, the effects of exercise on meat animals have not been conclusively investigated. Much of the literature on meat animals has been generated with little regard for the physiological basis of exercise, thus conclusions have been broadly interpreted to apply to any form of exercise. On the other hand, studies on the effects of exercise in humans and laboratory animals are physiologically based, with little emphasis on production or quality characteristics. This review of the literature will address the effects of exercise from a production perspective with emphasis on the effects of exercise on live animal performance, meat production and meat quality. In addition, information from studies on humans and laboratory animals will be included where appropriate. In some cases, their inclusion will be used to emphasize areas where production perspectives differ from physiological perspectives.

A. LIVE ANIMAL PERFORMANCE

Although the energy cost of exercise is relatively small compared to gross energy intake (Morrison *et al.*, 1968; Murray *et al.*, 1974a) the average daily gains of exercised farm animals are generally lower than in non-exercised animals (Mitchell and Hamilton, 1933; Bull and Rusk, 1942; Spaeth *et al.*, 1967; Morrison *et al.*, 1968). The lower average daily gains in exercised animals have been attributed to the combined effect of reduced feed intake and energy cost of the exercise (Crews *et al.*, 1969). Crews *et al.* (1969) also suggested that the degree of appetite suppression is due to the amount of stress associated with the exercise rather than due to the exercise *per se*. Surprisingly, most studies using pigs reported either no difference (Peo *et al.*, 1970; Mandigo *et al.*, 1971) or slightly higher (Murray *et al.*, 1974a) average daily gains in the exercised animals. Stevenson *et al.* (1966) studied the effect of different exercise regimes on feed intake in rats. They found that low levels of regular

exercise tended to decrease food intake whereas higher levels resulted in a proportionate increase in food intake. Perhaps the pigs were exercised sufficiently to produce an appetite stimulating effect since their feed intakes were not significantly different between exercised and control pigs.

B. MEAT PRODUCTION

Meat animal carcasses are composed mainly of muscle, fat and bone, with a small amount of anatomically distinct connective tissue. The most valuable component from a production standpoint is muscle. Thus, a considerable interest in altering the composition of an animal to produce more muscle has always existed in the field of animal science (Butterfield and Berg, 1974; Elsley, 1976; Allen, 1983; Price, 1986). For this reason, animal scientists have watched with curiosity the apparent success of weight training in turning the "90 pound weakling" into the perfect 'meat' animal; bulging hypertrophied muscles with low body fat.

Muscular hypertrophy

To properly discuss muscular hypertrophy it is necessary to define hypertrophy. For the purpose of this discussion, muscular hypertrophy is defined as an increase in an individual muscle weight as a percentage of total muscle weight, over and above normal growth patterns (ie. a muscle exists in a state not normally reached or passed through following normal growth processes). To benefit meat production, an increase in total muscle weight, or a shift in the distribution of muscle to the higher priced cuts must occur.

Unfortunately, besides common weight-lifting lore, there is very limited evidence to support the phenomenon of extensive muscular hypertrophy through exercise. Many authors have propagated this lore through unsubstantiated statements such as "while it is relatively simple to achieve muscle hypertrophy in man" (Macková and Hník, 1973) and "human muscle increases in size in response to exercise" (Gonyea and Ericson, 1976). Fox and Mathews (1981) state that "muscle enlargement with a corresponding increase in strength is a

commonly observed phenomenon"; they imply that muscular enlargement is visually apparent in humans when comparing flexed bicep muscles. McMorris and Elkins (1954) measured the circumference of human arms before and after twelve weeks of exercise and found slight increases in circumference, but were surprised that the increases were smaller than they would have expected from the appearance of the arm.

Historically, visual appraisal of beef animals has dominated carcass evaluation (Kempster *et al.*, 1982). The weaknesses of judging body composition entirely through visual appraisal were not shown until detailed compositional studies involving tissue separation were performed (Berg and Butterfield, 1976). In fact, human subjects offer little validation of muscular hypertrophy since most of the techniques used are, of necessity, non-invasive. In addition, studies with humans often involve comparisons between athletes and non-athletes (Katch *et al.*, 1980; Schantz *et al.*, 1981) rather than comparisons between the same subject before and after a training program. Comparisons between athletes and non-athletes are of questionable value as a basis for the occurrence of muscular hypertrophy since the subjects have genetically dissimilar backgrounds and they are not compared at the same body weight or at the same fat weight.

The common techniques for judging muscular hypertrophy in humans are through measurements of circumference (McMorris and Elkins, 1954) combined with skinfold thickness (Katch *et al.*, 1980), muscle cross-sectional area using tomography (Schantz *et al.*, 1981) and muscle fiber areas from muscle biopsies (MacDougall *et al.*, 1979; Schantz *et al.*, 1981); these techniques all have limitations. Measurements of muscular hypertrophy using circumference can be influenced by the thickness of subcutaneous tissue, the state of hydration, the amount of vasodilation, the length of the muscle at the time of measurement and the state of development of other included muscles (McMorris and Elkins, 1954). Similarly, skinfold thickness can be influenced by the amount of subcutaneous fat and the tonus of the underlying muscle. Any measurement of muscle fiber area or diameter should be accompanied by a measurement of sarcomere length (Swatland, 1980) since muscle fibers are considered to be isovolumetric (changes in sarcomere lengths can influence fiber diameters).

For example, Goldspink (1964) reported that the weights of exercised muscles were not greater than the weights of control muscles in mice, even though the fibers of the exercised muscles were of considerably larger diameter than fibers from control muscles. Although the use of tomography improves the measurement of muscle cross-sectional area, adjustments should still be made for the degree of contraction (average sarcomere length).

Documentation of muscular hypertrophy through exercise in laboratory animals has been even more limited than in humans. Many authors (Gutmann and Hájek, 1971; Macková and Hník, 1973; Gonyea and Ericson, 1976) attribute the development of a model of compensatory hypertrophy, through tenotomy of synergistic muscles (Denny-Brown, 1960), to the fact that muscular hypertrophy through exercise was so hard to produce in laboratory animals. This model not only produced the desired hypertrophy (albeit from passive mechanical factors rather than from increased phasic activity) but also inspired other models of hypertrophy: excision of synergistic muscles (Reitsma, 1970; Schiaffino and Pieroban Bormioli, 1973; Baldwin *et al.*, 1977), chronic muscle stretching (Sola *et al.*, 1973; Holly *et al.*, 1980; Ashmore and Summers, 1981) and partial paralysis of the hindlimb (Van Linge, 1962).

Compensatory hypertrophy resulting from artificial stresses is not directly comparable to hypertrophy potentially developing from exercise. Gutmann and Hájek (1971) and Gutmann *et al.* (1971) agreed that compensatory hypertrophy was of doubtful adaptive value and was not the same thing as hypertrophy through exercise. Macková and Hník (1973) found that compensatory hypertrophy was transient in character and was not substantially augmented by concomitant training. However, Ianuzzo and Chen (1979) claimed that the steady-state reached after the compensatory period was equivalent to true working hypertrophy. Goldberg *et al.* (1975) suggested that the rapid growth and partial regression in compensatory hypertrophy may be analogous to the over-compensation of most feedback devices or may result from the regrowth of the cut tendons. Gollnick *et al.* (1981) criticized compensatory hypertrophy as being non-physiological since it produces a 24 hour stretch on the muscle rather than the intermittent high tensions produced by activities such as weight-lifting. Gutman and Hájek (1971) compared increased phasic activity (through a

swimming exercise) to compensatory hypertrophy in rats. Increased phasic activity resulted in an increase of contractile proteins and a decrease in sarcoplasmic proteins; the opposite occurred in compensatory hypertrophy. In addition, they were unable to produce any hypertrophy through increased phasic activity. These models of compensatory hypertrophy have produced a wealth of information on the effects of tenotomy, excision of synergists, chronic muscle stretching and partial paralysis but have produced little information on muscular hypertrophy through exercise (Macková and Hník, 1973; Gonyea and Ericson, 1976). In any case, increased production through compensatory hypertrophy would be of little practical benefit to farm animal production systems.

Although hypertrophy through exercise has occasionally been claimed, close examination of these studies shows that the results are often inconclusive. Saville and Smith (1966) claimed to have induced muscular hypertrophy in exercised bipedal rats. However, their basis for demonstrating hypertrophy was through an increased ratio of hind leg muscle weight to body weight in bipedal rats compared to control rats. They made no adjustment for the lack of forelimbs in the bipedal rats; the effect may have been due to lower body weights, rather than increased muscle weights. Saville and Whyte (1969) also claimed muscular hypertrophy in rats engaged in a running exercise. Although the regression of muscle weight on body weight was significantly higher in exercised rats, there was no measurement of total body fat. Running exercise might be expected to reduce total body fat, resulting in higher muscle weight on body weight regressions in the exercised rats.

Two reports in the literature suggest that exercise-induced hypertrophy can occur in laboratory animals. Gonyea and Ericson (1976) developed a single-limb, weight-lifting model of hypertrophy in cats and compared contralateral limbs within an animal and between control and exercised animals. A within animal comparison successfully eliminates the need to take changes in total muscle mass into account. They found no significant differences in muscle weight or fiber diameters in control cats. In exercised cats, they reported an increase in the muscle weight and an increase in fiber diameters of the exercised limb. They did not adjust fiber diameters to a constant sarcomere length. Ho *et al.* (1980) successfully produced

hypertrophy of the *adductor longus* muscle, but failed to produce hypertrophy in the *rectus femoris* muscle in weight-lifting rats. These experiments indicate that exercise-induced changes in muscle distribution can occur, however there is little evidence that total muscle weight can increase.

Clearly, the measurement of muscular hypertrophy is not a simple one. Measurements of muscle circumference, fiber diameters, and muscle weights (with no reference to total muscle weight) are not useful predictors of hypertrophy from a production point of view.

Farm animals are useful for studies of muscular hypertrophy because, due to their size, full muscle dissections can be performed with relative ease to obtain total muscle weight and individual muscle weights. There is also a fairly extensive body of literature on muscle growth patterns in cattle (Butterfield and Berg, 1966b; Berg and Mukhoty, 1970; Butterfield and Johnson, 1971; Berg *et al.*, 1978), sheep (Lohse *et al.*, 1971; Butterfield *et al.*, 1984), and pigs (Richmond and Berg, 1971). However, even with the ability to do full dissections, there are still some problems with the use of farm animals. Firstly, inter-animal variation, although small, can mask increases in muscle weight, unless the changes are quite large. Gollnick *et al.* (1981) suggested that inter-animal comparisons should not be used due to the magnitude of inter-animal variation in muscle weights in rats. The large inter-animal variations they observed were probably due to comparisons at different total muscle weights. Secondly, it is necessary to make comparisons at the same total muscle weight. It is difficult to slaughter animals at the same total muscle mass; the same live weight does not ensure the same total muscle mass since differences in gut fill, offal components, and bone and fat weights can affect total muscle mass (Berg and Butterfield, 1976). Adjusting the data to similar total muscle mass can result in apparent changes in muscle distribution, since not all muscles grow at the same rate as total muscle mass (Dinkel *et al.*, 1965; Berg and Butterfield, 1966).

Despite these problems, several researchers have exercised farm animals, either in an attempt to increase total muscle mass or to change the distribution of muscle to an

economically more favorable distribution. Mitchell and Hamilton (1933) postulated, through indirect measurements, that muscular hypertrophy had occurred in steers exercised on a treadmill, although they did not measure hypertrophy *per se*. Bull and Rusk (1942) also exercised steers on a treadmill and obtained slightly higher dressing percentages and higher carcass grades in heavily exercised steers. They did not do full dissections to determine the nature of these changes at the tissue level. Skjervold *et al.* (1963) fed pigs from elevated feeders (forcing them to stand on their hind legs) to try to increase the amount of muscle in the ham. They found no effect on total muscle, or on the "meatiness" of commercial cuts. Spaeth *et al.* (1967) found no difference between treadmill-exercised sheep and controls in *longissimus* area, conformation grade or muscle fiber diameter. Peo *et al.* (1970) claimed a change in percent lean cuts (which could have been due to reduced fat or increased muscle) between treadmill-exercised and control pigs, but failed to report in which direction the change had occurred. Mandigo *et al.* (1971) observed no differences between control and treadmill-exercised pigs in percent lean cuts, percent ham and loin, or loin eye area. Murray *et al.* (1974a) concluded that treadmill exercise of pigs was ineffective in altering either the proportion of carcass muscle or its distribution. Butterfield (cited in Berg and Butterfield, 1976) found no difference in the amount of muscle or muscle weight distribution between exercised (hurdle jumping) and non-exercised calves.

Most of these researchers concluded that the intensity of the exercise was not sufficient to produce muscular hypertrophy (Skjervold *et al.*, 1963; Morrison *et al.*, 1968; Murray *et al.*, 1974a; Berg and Butterfield, 1976). However, intensity refers not only to the length of time that an exercise is performed, but also to the type of exercise. Exercise physiologists generally recognize two exercise extremes: (1) high intensity, short duration exercise (2) low intensity, prolonged duration exercise (Holloszy, 1967; Herbison and Gordon, 1973; Jaweed *et al.*, 1974). Furthermore, the level of intensity can be obtained by manipulation of resistance and velocity. Adaptational responses to either type of exercise vary, but it is the former type of exercise which is thought capable of producing muscular hypertrophy. Hence, most of the treadmill exercise routines used on farm animals would not

have been expected to produce muscular hypertrophy.

The only successful attempts to change muscle distribution in farm animals through exercise have used a chronic increase in the load carried by the abdominal muscles (pregnancy - Heap and Lodge, 1967; lead weights Lohse *et al.*, 1971 - as cited in Berg and Butterfield, 1976; fibrous feed vs. milk - Butterfield and Johnson, 1971). However, as Berg and Butterfield (1976) pointed out, the increase in the weight of the abdominal muscle, although due to physical stimulation, may not have been any larger than the muscle's genetic potential; the muscle did not reach a size greater than it would normally have reached at some point during its growth. Other researchers (Donaldson, 1935; Malina, 1969) have proposed that exercise accelerates growth of actively growing tissues although not necessarily past the tissue's mature weight or distribution. Evidence supporting this includes the fact that young actively growing animals are much more susceptible to the effects of exercise than mature animals (Malina, 1969; Macková and Hník, 1972; Goldspink and Howells, 1974; Goldberg *et al.*, 1975; Martin *et al.*, 1981).

Fat content of the carcass

Exercise, especially endurance exercise, has been reported to reduce total body fat in adult humans (Wilmore *et al.*, 1970; Boileau *et al.*, 1971). Although most farm animal studies involved a form of endurance exercise, researchers reported: lower yields of excess fat trim in exercised lambs (Spaeth *et al.*, 1967), significantly greater backfat depth in exercised pigs (Mandigo *et al.*, 1971), and no difference in the proportion of total fat between exercised and control pigs (Murray *et al.*, 1974a). Skjervold *et al.* (1963) found an increase in caul fat in pigs exercised by standing on their hind legs while feeding from elevated feeders. It is possible that the pigs in the experiments of both Skjervold *et al.* (1963) and Mandigo *et al.* (1971) had reached a stage at which fat was growing rapidly (slaughtered at approximately 90 kg). Thus, if exercise has an accelerating effect on tissue growth, we might expect to see increased fat in exercising pigs that had reached a fattening stage. Murray *et al.* (1974a) slaughtered their pigs at approximately 60 kg. The apparent contradiction between human

and farm animal studies may be due to the age of the subject. Human subjects are normally adults whereas farm animals are usually actively growing.

Bone content of the carcass

Few researchers working with farm animals have looked at the effects of exercise on bone growth. Skjervold *et al.* (1963) reported a decrease in length of the metatarsal and phalangeal bones in the hind limbs of pigs forced to feed from elevated feeders. Tulloh and Romberg (1963) found an increase in the width of the distal head of the metacarpal in load-carrying sheep. Anderson *et al.* (1971) demonstrated a conservation of bone in treadmill-exercised pigs.

There is a great deal more information on the effects of exercise on bone growth in the medical literature. Mechanical stresses and strains are considered essential for normal bone growth (Malina, 1969). However, both compressional and tensile forces generated through exercise have been reported to affect bone growth (Malina, 1969; Kiiskinen, 1977; Goss, 1978). Exercise has been shown to result in heavier (Saville and Smith, 1966; Anderson *et al.*, 1971; Souminen *et al.* 1980), thicker (Tulloh and Romberg, 1963; Malina, 1969) and shorter bones (Kiiskinen, 1977) with more mineral matter and less water content (Martin *et al.*, 1981). Malina (1969) attributed the increase in thickness to the increase in muscular tensions which serve as a stimulus to lateral growth. Bassett and Becker (1962) and Lakes and Saha (1979) presented the hypothesis that bone cells were able to respond to electric potentials generated by muscular tensions or compressional loads by increasing the amount of bone deposited. Malina (1969) also attributed the decrease in bone length to the growth-retarding compressive forces (as in a load-carrying exercise). Kiiskinen (1977) suggested that the decrease in bone length in treadmill-exercised mice may be due to an earlier closing of the epiphyses caused by the increased release of growth hormone induced by physical exercise.

The response of bone to exercise differs depending on the stage of maturity of the animal involved. Changes in bone shape induced by exercise seem to occur only in young

growing animals since the tibias of adult load-carrying beagles exhibited no change in shape (Martin *et al.*, 1981). Increased mineral content appears to occur in either growing animals (Anderson *et al.*, 1971) or adult animals (Martin *et al.*, 1981). Kiiskinen (1977) reported that increased bone weights in mice, resulting from treadmill running, were transient and disappeared when the mice reached maturity. He considered this to be an indication of accelerated bone growth. Another group of researchers (Bikle *et al.*, 1987) indicated that reduced activity in the growing rat resulted in reduced bone formation and retarded mineralization, akin to a decrease in mature bone.

Connective tissue in the carcass

Exercise has been shown to have significant effects on connective tissue (ligaments and tendons) in laboratory animals. These tissues have received very little attention in farm animal studies since they comprise a very small proportion of the carcass (approximately 1.0%). Tipton *et al.* (1975) reported an increase in ligament weight, width and thickness in exercised rats. Kiiskinen (1977) found that moderate training produced a transient increase in the dry weight of the Achilles tendon in treadmill-exercised mice. Again, Kiiskinen (1977) found that this effect disappeared when the mice reached maturity, implying that there was an acceleration of tendon growth due to exercise. Souminen *et al.* (1980) also suggested that the metabolism of collagen was accelerated by physical training in rapidly growing mice.

C. MEAT QUALITY

Meat quality is often neglected for the sake of meat production. However, meat quality is of much more value to the consumer than the amount of meat obtained per carcass. Since the effects of exercise are manifested at the cellular level first, with expression at the tissue level secondarily, it is reasonable to suspect exercise to affect meat quality.

Connective tissue

Exercise-related changes in connective tissue are of particular concern because meat quality is affected by the connective tissue content of muscle (Hiner *et al.*, 1955; Cross *et al.*, 1973; Dutson *et al.*, 1976). Studies of compensatory hypertrophy indicated large increases in the connective tissue content of muscle. Van Linge (1962) reported an enormous increase in connective tissue growing between hypertrophied muscle fibers in mice. Reitsma (1970) reported that hypertrophied muscles from rats were "hard from the great amount of connective tissue". Ianuzzo and Chen (1979) removed an excessive amount of connective tissue from hypertrophied rat muscles and Sparrow (1982) measured an increase in total collagen during hypertrophy in chickens, which he attributed to equal increases in both the epimysial collagen and the internal collagen (perimysial and endomysial) of the muscle. Goldberg *et al.* (1975) suggested that the proliferation of fibroblasts during compensatory hypertrophy results in an increase in the collagenous matrix surrounding the muscle cells and that this matrix may be involved in the transmission of contractile force.

Exercise is commonly believed to decrease meat tenderness, since range cattle and farm-yard hens traditionally had tougher meat than their confinement reared counterparts. However, the increased meat toughness in these animals is just as likely to be caused by a later stage of maturity at slaughter, or differences in intramuscular fat content. In addition, many authors (Hiner *et al.*, 1955; Smith and Carpenter, 1970; Kruggel and Field, 1974) have found more collagen in the so-called more "active" muscles than in the "less-active" muscles (eg. muscles in the distal limb vs. muscles in the proximal limb). Although the larger amounts of collagen in these muscles may be considered an adaptation to locomotion, it is misleading to believe that the increased collagen was due to more exercise. In fact, Kruggel and Field (1974) have indicated that collagen from the muscles in the "active" foreshank has less cross-linking than collagen from the "quiescent" *longissimus* muscle.

Contrary to common beliefs and to the results of experiments using models of compensatory hypertrophy, researchers studying exercise in farm animals have generally found decreases in connective tissue with a corresponding increase in meat tenderness. Mitchell and

Hamilton (1933) reported a decrease in the collagen content (expressed as a percentage of collagen nitrogen on total nitrogen) of the *biceps femoris* and *longissimus* muscles in treadmill-exercised steers. In addition, Mitchell and Hamilton (1933) found an increase in tenderness of cooked roasts and round steaks from exercised steers and postulated that the increase in tenderness was due to hypertrophy of muscle tissue with no corresponding increase in the connective tissue element. Other researchers have demonstrated an increase in myofibrillar protein as a result of exercise (Helander, 1961; Goldspink, 1964). Bull and Rusk (1942) noted a decrease in average resistance to shear of meat from ribs of exercised steers, with corresponding improved scores from taste panels. Decreased shear values have been significantly related to increased tenderness (Khan *et al.*, 1973). More recently, Spaeth *et al.* (1967) reported a significant increase in tenderness of meat from exercised lambs. However, Mandigo *et al.* (1971) and Hawrysh *et al.* (1974) found no difference in Warner-Bratzler shear (WBS) values of meat from exercised and control pigs. Hawrysh *et al.* (1974) performed the shear test on samples from the *longissimus*, which is not heavily involved in treadmill walking. Mandigo *et al.* (1971) did not report which muscle they used for their WBS measurements.

Fiber diameters and sarcomere lengths

Muscle fiber diameters contribute to meat tenderness and meat texture and have been found to change as a result of exercise. Muscle fiber diameters increased in exercised rats (Goldberg, 1967), cats (Gonyea and Ericson, 1976; Gonyea *et al.*, 1977) and humans (Schantz *et al.*, 1981). Larger fiber diameters and shorter sarcomere lengths are both correlated with decreased meat tenderness (Tuma *et al.* 1962; Bailey, 1972; Bouton *et al.*, 1975). Thus, regardless of whether increased fiber diameters as a result of exercise are real or due to shorter sarcomere lengths (Swatland, 1980), they could contribute to decreased meat tenderness.

Fiber types

Exercise has been shown to result in important shifts in the distribution of muscle fiber types. Three types of muscle fibers are generally recognized on the basis of their enzyme activities (Brooke and Kaiser, 1970; Essén *et al.*, 1975; Tunell and Hart, 1977), although some researchers believe that the population of fiber types is a continuum (Kowalski *et al.*, 1969; Guth and Yellin, 1971). Long-term endurance exercise increases oxidative enzyme activities (Essén-Gustavsson and Lindholm, 1985), decreases glycolytic enzyme activities (Green *et al.*, 1984) and exchanges fast-type for slow-type myosin isoforms (Pette, 1984). These changes result in a transition in fiber type from fast-oxidative-glycolytic (FOG) to slow-oxidative (SO). Green *et al.* (1984) also reported shifts from fast-glycolytic fibers (FG) to FOG fibers. Compensatory hypertrophy results in similar shifts in fiber type towards more SO fibers (Ianuzzo *et al.*, 1976; Baldwin *et al.*, 1976). Exercise of short duration and high intensity has been found to result in conversion from FOG fibers to FG fibers despite slowing of the contractile properties (Gonyea and Bonde-Petersen, 1978). Humans performing a weight-lifting exercise show a selective involvement of FG fibers; this supports a shift from FOG fibers to FG fibers during exercise of short duration and high intensity (Gollnick *et al.*, 1973). Other researchers reported conversion of FOG fibers to SO fibers (Jaweed *et al.*, 1977), or no change in fiber type (Thorstensson *et al.*, 1975, 1976). These responses are probably due to exercise intensities lower than those required for conversion of FOG fibers to FG fibers.

The properties of a muscle are thought to be a reflection of the muscle fiber types present (Cassens, 1977). Several researchers have attempted to relate muscle fiber types to meat quality. May *et al.* (1977) attempted to relate differences in fiber type composition in several breeds of cattle to measurements of WBS and taste panel scores on tenderness, flavour, juiciness and over-all acceptability. They found no relation between fiber type composition and any of the palatability traits measured. However, Melton *et al.* (1974) found a positive correlation between taste panel flavour scores and areas of intermediate fibers (FOG) and white fibers (FG) in cattle. Similarly, Seideman and Theer (1986) found

that the percentage area of white fibers (FG) was positively correlated with tenderness and that percentage area of intermediate fibers was negatively correlated with tenderness. In contrast, Moody *et al.* (1980) reported a significant correlation between red fiber diameters (SO) and tenderness. Thus, shifts in fiber type resulting from exercise may affect meat quality.

Subcellular changes

In addition, exercise may result in shifts in enzyme activity, changes in energy substrates and alterations in buffering capacity, all of which affect post-mortem glycolysis. Changes in the rate or extent of post-mortem glycolysis alters post-mortem pH decline and ultimate pH, both of which are closely related to meat quality (Dutson, 1983). Post-mortem pH decline and ultimate pH have been associated with water-holding capacity (McClain and Mullins, 1969; Bouton *et al.*, 1972b), tenderness (Bouton *et al.* 1972a; Yu and Lee, 1986), and sarcomere length (Jeremiah, 1978).

Exercise related changes in enzyme activities may influence meat quality. Hamm (1977) indicated that phosphofructokinase (PFK) and phosphorylase are the rate limiting enzymes in post-mortem glycolysis. Phosphofructokinase activity has been shown to increase as a result of long-term endurance exercise (Gollnick *et al.*, 1973). Baldwin and co-workers (1976, 1977) report decreased activities of PFK and citrate synthase (CS) during compensatory hypertrophy. Ianuzzo and Chen (1979) interpret these findings as merely a dilution effect resulting from a selective lag in protein synthesis of these enzymes during the acute stage of hypertrophy. During the steady state of compensatory hypertrophy, normal PFK and CS activities exist. Enzyme activity of myokinase was increased in sprint-trained and strength-trained men (Thorstensson *et al.*, 1975, 1976). Creatine phosphokinase (CPK) activity was increased in sprint trained men (Thorstensson *et al.*, 1975). Both of these enzymes contribute to the maintenance of ATP levels during the post-mortem delay phase (Bendall, 1973). Martin *et al.* (1977) reported that CPK levels were related in a non-linear fashion to muscle color and tenderness.

Exercise can alter energy substrate levels which are related to meat tenderness and meat colour. Gollnick and co-workers (1972, 1973) found higher glycogen concentrations in athletes vs. untrained subjects and observed a 250% increase in glycogen after, as compared to before training. Karlsson *et al.* (1972) reported an increase in resting ATP concentrations with training. Ultimate pH has been shown to vary inversely with glycogen reserves (Bendall, 1973). Pattengale and Holloszy (1967) confirmed the work of Whipple (1926) that prolonged endurance exercise increased the concentration of muscle myoglobin. Myoglobin is the major pigment involved in the development of meat colour (Forrest *et al.*, 1975).

Finally, exercise has been shown to affect the buffering capacity of a muscle, which may alter post-mortem pH decline, and the resulting tenderness of the meat. Buffering capacity was higher in anaerobically trained athletes (McKenzie *et al.*, 1983). Hodgson (1985) suggested that it may be possible to increase the buffering capacity of muscle with training. Carnosine, one of the major components involved in post-mortem muscle buffering capacity between pH 7.00 and pH 5.5 (Bate-Smith, 1938), has been demonstrated to increase in anaerobically trained humans (Parkhouse *et al.*, 1983). There is still some question as to whether or not carnosine's function in living muscle is that of a physiological buffer (Bate-Smith, 1938; Parkhouse *et al.*, 1983). In any case, changes in buffering capacity would affect the post-mortem pH decline and may thus affect meat quality.

D. CONCLUSION

Clearly exercise affects many body systems at many levels, depending on the intensity and duration of the exercise. Animal scientists are primarily interested in the effects of exercise on live animal performance, meat production and meat quality.

The intensity of an exercise appears to have a major effect on live animal performance. Low intensity exercise results in lowered feed consumptions and decreased average daily gains. High intensity exercise results in increased feed consumptions and little difference in average daily gains. Pigs may be an exception to other farm animals, since even relatively low levels of exercise appear to stimulate appetite.

Meat production can be influenced through changes to any of the major carcass tissues: muscle, fat and bone. Despite a common belief that exercise can increase total muscle, there is little evidence that this occurs. Exercise has been shown to decrease fat in adult humans and to increase the deposition of bone. However, most of the changes induced by exercise appear to be age-related. Many of the discrepancies in the literature may result from comparisons between animals at different stages of maturity.

A number of studies have shown that exercise increases meat tenderness. The mechanism causing this is not known. However, exercise does appear to affect many contributors to meat tenderness, including connective tissue metabolism, enzyme activity, energy stores and buffering capacity.

A major question addressed in this thesis is whether muscular hypertrophy actually occurs as a result of exercise. The first three experiments in the following thesis (Chapters II to IV) were designed to address this question and involved short-term, high intensity exercises. Since little information on this type of exercise has been published, additional information on the effects of these exercises on feed intake, body composition and meat quality was collected. The fourth experiment (Chapter V) was designed to study the effects of a long-term, endurance exercise on composition at the gross and cellular level and to relate these changes to meat quality. A concluding chapter (Chapter VI) reiterates and integrates the important conclusions from the previous chapters and discusses their implications to exercise physiology and animal science.

Chapter II

THE EFFECTS OF STATIC LOAD-CARRYING ON GROWTH AND DEVELOPMENT OF SHEEP

A. INTRODUCTION

Many researchers have suggested that a main factor governing total muscle weight in an animal is the load that it has to support (Bryden, 1969; Berg and Butterfield, 1976; Price, 1976). In most cases, this load would be the live weight of the animal. In theory, as live weight increases, the total muscle mass required to support it must also increase. In addition, muscle distribution at any total muscle weight is thought to be relatively fixed within a species (Berg and Butterfield, 1976). However, it is generally believed among physiologists that individual muscles are capable of responding to increased functional demands through hypertrophy (Goldberg *et al.*, 1975; Goldspink, 1977; Goss, 1978; Gollnick *et al.*, 1981) or hyperplasia (Sola *et al.*, 1973; Gonyea *et al.*, 1977; Gonyea, 1980; Ho *et al.*, 1980). This would result in either an increase in total muscle mass or a change in muscle distribution.

The following pilot study was designed to artificially increase the live weight of sheep through load-carrying and to monitor the effects on total muscle weight and muscle weight distribution.

B. MATERIALS AND METHODS

Six shorn Suffolk-cross wether lambs averaging 28 kg were assigned to this experiment. All sheep were penned individually and were fed the same basic diet of 50% alfalfa pellets and 50% barley-based concentrate (Table II.1). The sheep were fitted with modified canvas show blankets (Plate II.1) which were worn continuously for the duration of the experiment. The sheep were randomly allocated to three treatments: Treatment I (Maintenance) sheep were fed to maintain a constant live weight; Treatment II (Growing) sheep were fed to gain weight (approximately 0.2 kg per day); Treatment III (Load-carrying) sheep were fed to maintain constant live weight but carried lead weights in the show blankets

Table II.1 The formulation of the pelleted diet which was fed to maintenance, growing and load-carrying sheep.

Composition (g.kg^{-1}) [†]		Analysis (dry matter basis, g.kg^{-1})	
Alfalfa pellets	500	Crude protein	165
Barley	278	Calcium	13.1
Oats	57	Phosphorous	3.0
Soybean meal	83		
Alfalfa meal	57	Digestible energy, MJ.kg^{-1}	12.13
Molasses	13		

[†] To 100 kg of this diet were added 700 g salt, 700 g ground limestone and 28 g Vitamin A-D-E (10,000,000 i.u./kg Vit A, 1,650,000 i.u./kg Vit D, 10,000 i.u./kg Vit E).



Plate II.1 The modified canvas sheep show blanket

to simulate the live weight gain of the sheep in Treatment II (Figure II.1).

Daily feed consumption and live weight were recorded. After eight weeks on test the sheep in Treatment III were carrying weight in the blankets amounting to 43% of their own live weight. At that time, a video recorder was set up for 10 days to monitor any differences in activity patterns among the treatments. Activities were classified into resting (lying down) or active (moving, standing, eating) categories. Animals were monitored for four 15 minute periods starting at 6:00 a.m., 12:00 noon, 6:00 p.m. and 12:00 midnight for a total of 1 hour recording time per animal. The sheep were on experiment for 16 weeks.

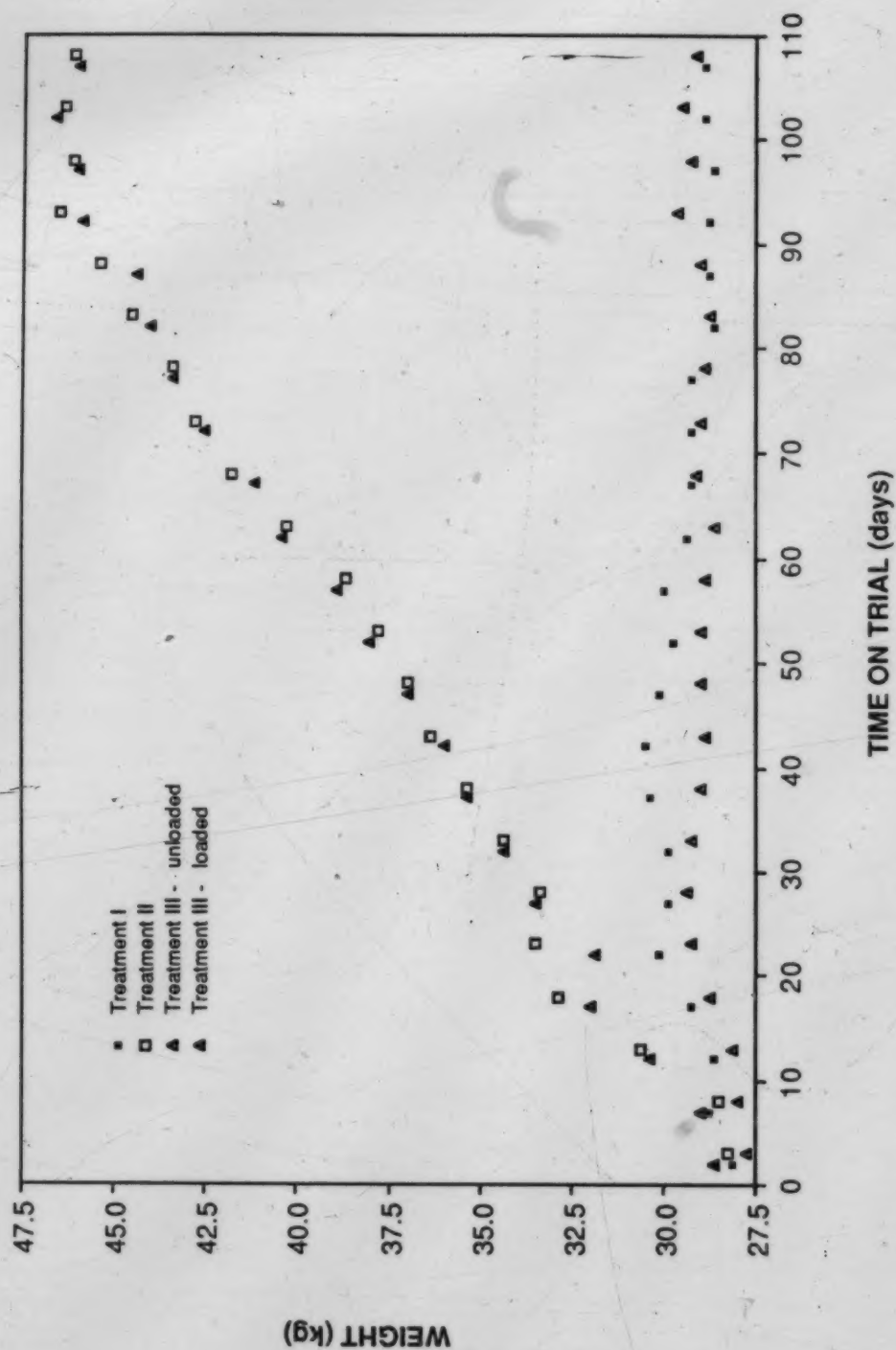
At the end of the experiment the sheep were 'shrunk' for 24 hours (free access to water but no feed), stunned with a captive bolt pistol and killed by jugular/carotid exsanguination. The bodies were dressed in the normal commercial manner and the following offal components were weighed: the head, disarticulated between the occipital bone and the atlas vertebra; the fleece; the front feet, disarticulated distally to the carpals; the hind feet, disarticulated distally to the tarsals; the empty heart (trimmed of fat); the lungs; the liver (gallbladder removed); the kidneys (trimmed of fat); the spleen; the empty bladder; and the penis.

The carcasses were then split medially and 1.25 cm thick chops were cut from the *longissimus* at the level of the last rib. These samples were kept on ice until they could be processed for histological staining.

The split carcasses were chilled for 48 hours at 6° C. The chilled left sides were completely dissected to obtain individual muscle and individual bone weights (Butterfield and May, 1966). Body fat was separated into subcutaneous, intermuscular and perirenal plus channel fat depots and weighed. Connective tissue (including tendons severed at the point where muscle tissue began, small pieces of cartilage removed from the joints, and pieces of fascia) was weighed. The weight of the *ligamentum nuchae* was recorded separately.

The samples taken for histological examination were subsampled and mounted on cryostat chucks by freezing in isopentane cooled in liquid nitrogen. Ten micron sections were cut on the cryostat and stained for myosin ATPase activity (Guth and Samaha, 1970;

**Figure II.1 Five-day average live weights of maintenance, growing and load-carrying sheep
(offset for clarity)**



Appendix I.B).

Weights of individual muscles, bones and fat depots were expressed both as absolute values and as proportions of the appropriate carcass tissue weight. Total muscle, bone and fat weights, and offal components were expressed as absolute weights and as proportions of carcass weight. All data were analysed using a one-way analysis of variance to compare treatments. A Student-Neuman-Keuls multiple range test was used to separate treatment means which were significantly different at, or below, the 5% probability level (Steel and Torrie, 1980). Average daily gains were calculated using a regression of live weight with time on trial. SPSSX (1983) was used for statistical analyses.

C. RESULTS

Initial weights of sheep in all three treatments were similar by design (Table II.2). At the end of the experiment, the weights of the maintenance and load-carrying sheep were similar, also by design, and significantly different ($P < 0.01$) from the weights of the growing sheep in Treatment II. Figure II.1 shows the close relationship in five day average live weights between Treatment I and III throughout the experiment. The artificial weight gain (in the form of lead weights) of sheep in Treatment III closely paralleled the actual gain of sheep in Treatment II (Figure II.1).

Feed consumption was not *ad libitum* hence average daily gains and feed efficiency calculations were only meaningful for the growing sheep ($0.19 \text{ kg} \cdot \text{day}^{-1}$ and $4.1 \text{ kg feed} \cdot \text{kg gain}^{-1}$, respectively). The load-carrying sheep consumed only 9% more feed than the maintenance sheep (65.6 kg vs. 60.0 kg) to maintain their weight despite carrying loads equivalent to 60% of their live weight by the end of the experiment.

Total carcass weight (Table II.3) was significantly greater in the growing sheep than in the maintenance sheep (22.2 kg vs. 11.4 kg; $P < 0.01$). The head and the hind feet were significantly smaller as a proportion of total carcass weight in the growing sheep than in either the load-carrying or maintenance sheep. However, when expressed as absolute weights (Table II.4), the head and hind feet were slightly heavier in the growing sheep. The

Table II.2 Live animal performance of maintenance, growing and load-carrying sheep.

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Number of animals	2	2	2	—	—
Initial weight, kg	28.2	28.9	28.0	0.9	—
Final weight, kg	28.5 ^a	46.3 ^b	29.0 ^a	0.2	<0.01
Weight gain, kg	0.3 ^a	17.4 ^b	1.0 ^a	0.8	<0.01
Average daily gain, kg.day ⁻¹	<0.01	0.19	±0.01	0.02	—
Feed consumption, kg	60.0 ^a	157.8 ^b	65.6 ^a	3.4	<0.01
Feed conversion efficiency, kg.kg ⁻¹	—	4.1	—	0.3	—

^{a,b} Means not followed by the same letter are significantly different at the 5% level of probability.

Table II.3 Offal components (as a percentage of total carcass weight) of maintenance, growing and load-carrying sheep.

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Number of animals	2	2	2	—	—
Total carcass weight, kg	11.4 ^a	22.2 ^b	12.2 ^a	0.47	<0.01
Fleece	19.5	22.2	19.7	2.07	0.64
Head	16.3 ^a	11.1 ^b	16.8 ^a	1.01	0.05
Front feet	3.0 ^a	1.9 ^b	3.3 ^c	0.04	<0.01
Hind feet	3.0 ^a	1.9 ^b	2.7 ^a	0.16	0.03
Heart	1.2	0.8	1.0	0.11	0.16
Lungs	2.6	1.9	2.8	0.28	0.20
Liver	2.7 ^a	3.5 ^b	2.9 ^{a,b}	0.14	0.05
Kidneys	0.55	0.51	0.45	0.04	0.44
Spleen	0.31	0.39	0.36	0.06	0.70
Bladder	0.11	0.09	0.16	0.03	0.38
Penis	0.18	0.11	0.28	0.06	0.28

a,b,c Means not followed by the same letter are significantly different at the 5% level of probability.

Table II.4 Absolute weights of selected offal components from maintenance, growing and load-carrying sheep.

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Number of animals	2	2	2	—	—
Head, kg	1.86	2.46	2.03	0.11	0.06
Hind feet, kg	0.34	0.41	0.33	0.02	0.14
Front feet, kg	0.34 ^a	0.43 ^b	0.40 ^b	0.01	0.03
Liver, kg	0.30 ^a	0.78 ^b	0.35 ^a	0.04	<0.01

^{a, b} Means not followed by the same letter are significantly different at the 5% level of probability.

proportion of front feet (Table II.3) was significantly heavier in the load-carrying sheep (3.3%) than in the growing sheep (1.9%) or the maintenance sheep (3.0%). In absolute terms (Table II.4), the front feet from the load-carrying sheep were similar in weight to those from the growing sheep and were significantly heavier than the front feet from the maintenance sheep (0.40 vs. 0.34 kg; $P=0.03$). The proportion of liver (Table II.3) in the load-carrying sheep lay intermediate between the maintenance and growing sheep and was not significantly different from either group. The maintenance sheep had a significantly smaller proportion of liver than the growing sheep (2.7% vs. 3.5%). When expressed as an absolute weight (Table II.4), there was no difference in liver weight between the load-carrying and maintenance sheep.

The half carcass weights of Treatments I and III were similar and significantly different from those of the growing sheep (Table II.5). Weights of the major carcass tissues showed similar trends in that the maintenance and load-carrying sheep were similar and significantly different from the growing sheep. However, the percentage of connective tissue in the load-carrying animals was significantly larger than in either the maintenance or growing sheep (1.2 vs. 0.6, 0.8%, respectively).

A breakdown of connective tissue weights (Table II.6) showed that the growing sheep had significantly more miscellaneous connective tissue than either the maintenance or load-carrying sheep. However, the load-carrying sheep had significantly heavier ($P=0.02$) *ligamenta nuchae* than either the maintenance or growing sheep (37.5 vs. 14.0 and 15.0 g, respectively).

Analysis of the weight distribution among anatomical muscle groups (Figure II.2; Berg and Butterfield, 1976) revealed that muscle Groups 3 and 7 (muscles around the spinal column and muscles in the thorax to thoracic limb) were significantly heavier ($P=0.02$) in the growing sheep than in sheep of the other treatments (Table II.7). Muscle Group 6 (distal thoracic limb) tended to be smaller in the growing sheep than in the maintenance or load-carrying sheep. Although there were no significant differences at the 5% level between the load-carrying and maintenance sheep, some interesting changes occurred in muscle Group

Table II.5 Half carcass dissection of maintenance, growing and load-carrying sheep.

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Number of animals	2	2	2	—	—
Half carcass weight, kg	5.8 ^a	11.4 ^b	5.8 ^a	0.2	<0.01
Carcass [†]	39.9	48.1	41.9	1.5	0.06
Muscle [‡]	62.7	57.3	63.5	1.1	0.06
Fat [‡]	10.8 ^a	26.9 ^b	10.9 ^a	1.3	<0.01
Bone	22.8 ^a	13.9 ^b	21.5 ^a	0.5	<0.01
Connective tissue [‡]	0.6 ^a	0.8 ^a	1.2 ^b	<0.01	0.03
Muscle:bone ratio	2.8 ^a	4.1 ^b	3.0 ^a	0.1	<0.01

[†] Warm carcass weight expressed as a percentage of live weight.

[‡] Expressed as a percentage of half carcass weight.

a, b Means not followed by the same letter are significantly different at the 5% level of probability.

Table II.6 Connective tissue weights of maintenance, growing and load-carrying sheep.

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Number of animals	2	2	2	—	—
Miscellaneous connective tissue, g	23.0 ^a	74.0 ^b	32.5 ^a	4.0	<0.01
Ligamentum nuchae, g	14.0 ^a	15.0 ^a	37.5 ^b	3.1	0.02

^{a, b} Means not followed by the same letter are significantly different at the 5% level of probability.

Table II.7 Anatomical muscle group weights (as a percentage of half carcass muscle weight) of maintenance, growing and load-carrying sheep.

Muscle group	Treatment I Maintenance		Treatment II Growing		Treatment III Load-Carrying		S.E.M.	Probability
	2	3.61 ^a	2	6.54 ^b	2	3.71 ^a	0.14	— <0.01
Number of animals	2		2		2			
Half carcass muscle weight, kg		3.61 ^a		6.54 ^b		3.71 ^a	0.14	— <0.01
1. Proximal pelvic limb	30.4		27.6		29.2		0.71	0.15
2. Distal pelvic limb	5.5		4.4		5.0		0.22	0.86
3. Around spinal column	15.1 ^a		17.5 ^b		14.8 ^a		0.31	0.02
4. Abdominal wall	11.1		13.5		12.2		0.58	0.13
5. Proximal thoracic limb	13.3		12.8		15.2		0.45	0.07
6. Distal thoracic limb	3.2		2.9		3.3		0.09	0.07
7. Thorax to thoracic limb	6.6 ^a		8.2 ^b		6.7 ^a		0.20	0.02
8. Neck to thoracic limb	3.8		4.3		4.2		0.34	0.65
9. Neck and thorax	10.9		8.8		9.3		0.59	0.17

^{a, b} Means not followed by the same letter are significantly different at the 5% level of probability.

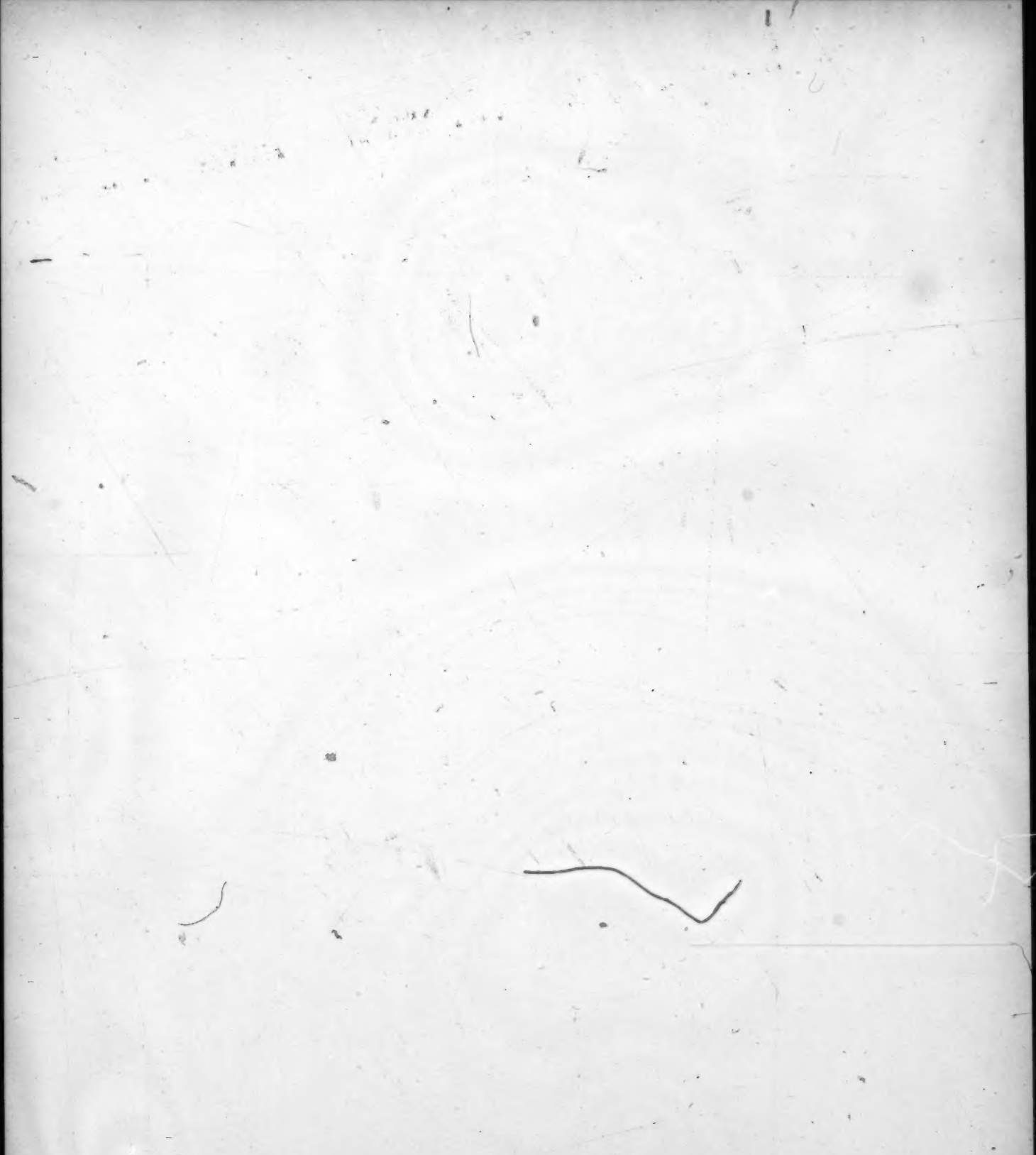
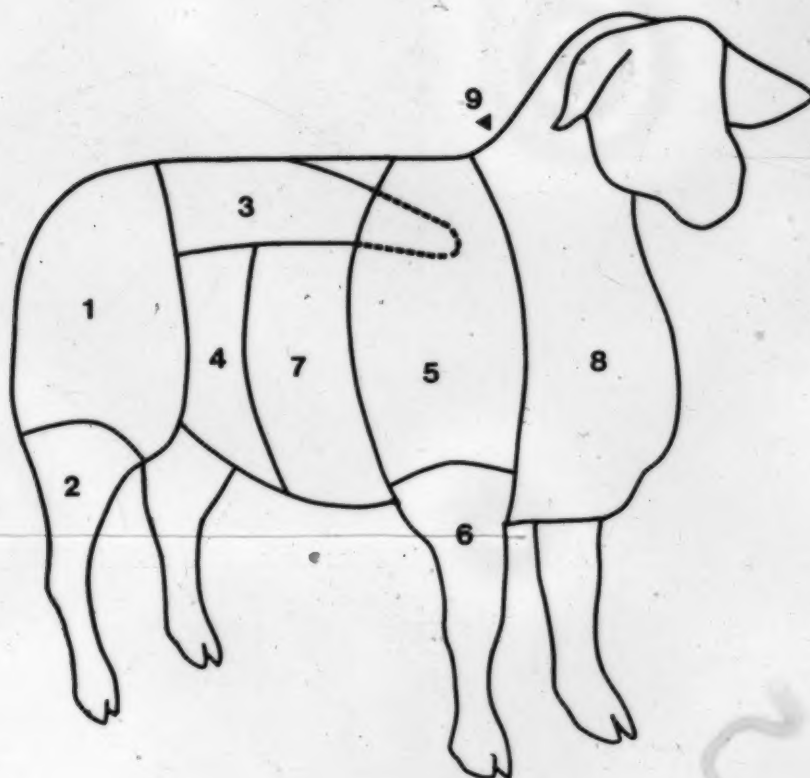


Figure II.2 The relative locations of the anatomical muscle groups in sheep



1. Proximal pelvic limb
2. Distal pelvic limb
3. Around spinal column
4. Abdominal wall
5. Proximal thoracic limb
6. Distal thoracic limb
7. Thorax to thoracic limb
8. Neck to thoracic limb
9. Neck and thorax

5, the proximal thoracic limb. Load-carrying animals tended toward ($P=0.07$) a higher proportion of muscle in this group than either the maintenance or growing animals (15.2 vs. 13.3 and 12.8%). Further analysis of this group (expressing individual muscles as a percentage of total muscle weight; Table II.8) showed that the *supraspinatus* was the only muscle percentage which was significantly larger at the 5% level in the load-carrying sheep. However, the proportions of several other muscles were slightly larger in the load-carrying sheep than in the maintenance sheep (*deltoideus*, *infraspinatus*, *triceps brachii* (*caput laterale* and *caput longum*), *biceps brachii*, *coracobrachialis*, *brachialis*, *brachiocephalicus*), contributing to the higher proportion of muscle in Group 5 in the load-carrying sheep.

Some of the muscles in Group 9 (neck and thorax) occupied significantly smaller proportions of total muscle mass in the load-carrying sheep than in the maintenance sheep: the *splenius*, the *longissimus capitus* and *atlantis* and the *longus colli*. Eight muscles out of 97 were similar between the load-carrying and the maintenance sheep and significantly different ($P<0.05$) from the growing sheep. In two muscles (*iliocostalis* and *semispinalis capitus*), the load-carrying sheep had the same proportion of these muscles as the growing sheep. The *iliocostalis* was larger in the load-carrying and growing sheep than in the maintenance sheep. The *semispinalis capitus* was smaller in the growing and load-carrying sheep than in the maintenance sheep.

Although the load-carrying sheep had the same weight of total bone as the maintenance sheep, the anatomical partitioning tended towards more bone weight in the appendicular skeleton rather than in the axial skeleton (Table II.9). The maintenance sheep had almost exactly the same percentage of bone in the axial and appendicular locations as the growing sheep (48.6, 51.3 vs. 48.2, 51.7%). The load-carrying sheep tended to have less bone in the axial location (45.2%) and more bone in the appendicular location (54.8%). Further analysis showed that the total thoracic leg bone weight (scapula, humerus, radio-ulna, and the carpals) in the load-carrying sheep was significantly heavier ($P<0.01$) than that in the maintenance sheep (259 vs. 233 g).

Table II.8 Muscle distribution (individual muscle weights as a percentage of half carcass muscle weight) of maintenance, growing and load-carrying sheep.

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Half carcass muscle weight, kg	3.61 ^a	6.54 ^b	3.71 ^a	0.14	<0.01
Muscle Group 1 - Proximal Pelvic Limb					
1. Tensor fascia latae	0.80	1.10	0.87	±0.106	0.27
2. Biceps femoris	5.14	5.09	4.95	±0.192	0.78
3. Gluteus medius	3.16	2.81	3.12	±0.184	0.44
4. Vastus lateralis	2.79	2.83	2.62	±0.261	0.84
5. Gluteus accessorius	0.59	0.55	0.36	±0.055	0.12
6. Gluteus profundus	0.47	0.27	0.39	±0.089	0.39
7. Rectus femoris	2.69	2.37	2.70	±0.070	0.07
8. Semitendinosus	1.89	1.86	1.96	±0.083	0.73
9. Gracilis	0.93	1.05	0.84	±0.116	0.52
10. Semimembranosus	5.50	4.41	5.08	±0.207	0.07
11. Adductor femoris	2.41 ^a	1.76 ^b	2.30 ^a	±0.112	0.05
12. Pectineus	0.57	0.56	0.55	±0.039	0.90
13. Sartorius	0.23	0.21	0.19	±0.043	0.86
14. Gemellus	0.16	0.05	0.07	±0.061	0.50
15. Quadratus femoris	0.58	0.47	0.56	±0.016	0.33
16. Obturator internus et externus	0.43	0.53	0.61	±0.083	0.43
17. Vastus medialis	0.93	0.75	0.92	±0.056	0.17
18. Vastus intermedius	0.99	0.82	0.92	±0.059	0.26
19. Articularis genu	0.07	0.10	0.10	±0.026	0.68
20. Iliacus	0.57	0.49	0.58	±0.126	0.85

Continued

Table II.8 (cont'd.)

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Muscle Group 2 - Distal Pelvic Limb					
1. <i>Gastrocnemius et soleus</i>	1.95 ^a	1.55 ^b	1.86 ^a	±0.057	0.03
2. <i>Flexor digitorum superficialis</i>	0.70	0.49	0.70	±0.058	0.14
3. <i>Extensor group</i>	0.76	0.60	0.57	±0.052	0.15
4. <i>Peroneus longus</i>	0.19	0.18	0.19	±0.012	0.74
5. <i>Extensor digiti quarti proprius</i>	0.23	0.19	0.12	±0.038	0.29
6. <i>Tibialis anterior</i>	0.24	0.19	0.19	±0.050	0.72
7. <i>Tibialis posterior</i>	0.21	0.13	0.26	±0.058	0.67
8. <i>Popliteus</i>	0.34	0.28	0.45	±0.071	0.36
9. <i>Flexor digitorum longus</i>	0.06	0.13	0.19	±0.047	0.58
10. <i>Flexor hallucis longus</i>	0.63	0.61	0.47	±0.152	0.74
Muscle Group 3 - Spinal					
1. <i>Psoas minor</i>	0.54	0.48	0.52	±0.045	0.65
2. <i>Psoas major</i>	1.56	1.53	1.45	±0.102	0.74
3. <i>Quadratus lumborum</i>	0.36	0.46	0.28	±0.108	0.57
4. <i>Iliocostalis</i>	0.56 ^a	0.40 ^b	0.39 ^b	±0.028	0.04
5. <i>Longissimus</i>	8.81	9.52	8.68	±0.161	0.06
6. <i>Spinalis dorsi</i>	1.42	1.63	1.33	±0.132	0.39
7. <i>Multifidus dorsi</i>	1.56 ^a	3.23 ^b	1.85 ^a	±0.089	<0.01
8. <i>Longissimus cervicis</i>	0.30	0.26	0.31	±0.052	0.80

Continued

Table II.8 (cont'd.)

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Muscle Group 4 - Abdominal					
1. Cutaneous trunci	0.75	2.40	1.61	±0.321	0.08
2. Serratus dorsalis caudalis	0.08	0.07	0.11	±0.051	0.83
3. Obliquus externus abdominis	1.62	2.11	1.73	±0.199	0.33
4. Retractor costae	0.10	0.09	0.12	±0.032	0.74
5. Obliquus internus abdominis	1.20	1.38	1.45	±0.197	0.69
6. Transversus abdominis	1.68	1.71	1.16	±0.248	0.34
7. Rectus abdominis	1.69	2.07	1.99	±0.256	0.60
8. Diaphragm	3.96	3.67	4.04	±0.111	0.19
Muscle Group 5 - Proximal Thoracic Limb					
1. Deltoideus	0.42	0.50	0.50	±0.024	0.14
2. Infraspinatus	2.31	2.34	2.63	±0.144	0.35
3. Triceps brachii (caput laterale)	0.89	0.80	0.95	±0.052	0.28
4. Teres minor	0.21	0.19	0.21	±0.019	0.63
5. Triceps brachii (caput longum)	2.85	2.57	3.12	±0.142	0.07
6. Tensor fasciae antibrachii	0.30	0.31	0.29	±0.019	0.87
7. Supraspinatus	2.25 ^a	2.17 ^a	2.71 ^b	±0.056	0.01
8. Biceps brachii	0.69	0.56	0.77	±0.053	0.14
9. Teres major	0.53	0.37	0.50	±0.112	0.60
10. Coracobrachialis	0.21	0.20	0.24	±0.019	0.40
11. Subscapularis	1.35 ^a	1.18 ^b	1.38 ^a	±0.031	0.04
12. Brachialis	0.39	0.46	0.55	±0.106	0.61
13. Brachiocephalicus	0.65	0.93	1.09	±0.276	0.58
14. Triceps brachii (caput mediale)	0.23	0.27	0.22	±0.070	0.88

Continued

Table II.6 (Cont'd.)

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Muscle Group 6 - Distal Thoracic Limb					
1. <i>Extensor carpi radialis</i>	0.82	0.82	0.84	±0.050	0.95
2. <i>Extensor digitorum longus</i>	0.13	0.13	0.14	±0.016	0.92
3. <i>Extensor digitorum communis</i>	0.10	0.13	0.16	±0.029	0.41
4. <i>Extensor digitorum lateralis</i>	0.10	0.12	0.07	±0.013	0.17
5. <i>Extensor carpi ulnaris lateralis</i>	0.47 ^a	0.36 ^b	0.47 ^a	±0.015	0.02
6. <i>Abductor pollicis longus</i>	0.08	0.04	0.03	±0.003	0.54
7. <i>Flexor carpi radialis</i>	0.13	0.12	0.19	±0.044	0.56
8. <i>Flexor carpi ulnaris</i>	0.20	0.25	0.25	±0.040	0.65
9. <i>Flexor digitorum sublimis</i>	0.62	0.39	0.40	±0.072	0.18
10. <i>Flexor digitorum profundus</i>	0.49	0.37	0.59	±0.158	0.66
11. <i>Anconaeus</i>	0.19	0.17	0.22	±0.029	0.54
Muscle Group 7 - Thorax to Thoracic Limb					
1. <i>Latissimus dorsi</i>	1.27 ^a	1.72 ^b	1.22 ^a	±0.046	<0.01
2. <i>Trapezius thoracic</i>	0.26	0.45	0.34	±0.078	0.33
3. <i>Serratus ventralis thoracis</i>	1.56	2.05	1.68	±0.144	0.19
4. <i>Pectoralis profundus</i>	2.60	2.92	2.47	±0.100	0.10
5. <i>Pectoralis superficialis</i>	0.92	1.01	0.96	±0.064	0.62

Continued

Table 11.8 (Cont'd.)

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Muscle Group 8 - Neck to Thoracic Limb					
1. Trapezius cervicalis	0.29	0.31	0.29	±0.020	0.79
2. Omotraversarius	0.32	0.42	0.20	±0.162	0.66
3. Rhomboides	0.40	0.69	0.74	±0.098	0.16
4. Serratus ventralis cervicis	2.81	2.84	2.97	±0.209	0.85
Muscle Group 9 - Neck and Thorax					
1. Intercostales	2.98	2.65	2.93	±0.149	0.36
2. Serratus dorsalis cranialis	0.06	0.04	0.19	±0.091	0.53
3. Scalenus dorsalis	0.07 ^a	0.13 ^b	0.03 ^a	±0.011	0.01
4. Cervicohyoideus	0.03	0.03	0.04	±0.011	0.73
5. Splenius	0.24 ^a	0.20 ^b	0.15 ^c	±0.009	0.01
6. Scalenus ventralis	0.41	0.56	0.49	±0.024	0.05
7. Longus capitis	0.26 ^a	0.15 ^b	0.24 ^a	±0.014	0.02
8. Intertransversarius longus	0.47	0.19	0.32	±0.088	0.22
9. Longissimus capitis and atlantis	0.52	0.41	0.28	±0.067	0.18
10. Intertransversarii cervicis	0.81	0.81	0.80	±0.248	0.99
11. Semispinalis capitis	0.83 ^a	1.19 ^b	1.06 ^b	±0.049	0.03
12. Obliquus capitis caudalis	1.00	0.16	0.54	±0.328	0.33
13. Rectus thoracis	0.14	0.03	0.04	±0.071	0.06
14. Transversus thoracis	0.24	0.29	0.33	±0.092	0.82
15. Longus colli	1.16 ^a	1.04 ^a	0.68 ^b	±0.057	0.02
16. Multifidi cervicis	0.99	0.48	0.58	±0.196	0.30
17. Sternocapalicus	0.64	0.42	0.60	±0.215	0.76

^{a,b,c} Means not followed by the same letter are significantly different at the 5% level of probability.

Table II.9 Anatomical partitioning of bone (as a percentage of half carcass bone) from maintenance, growing and load-carrying sheep.

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Number of animals	2	2	2	-	-
Absolute weight, g					
Axial skeleton	627	771 ^b	565	70.5	0.25
Appendicular skeleton	664 ^a	819 ^b	682 ^a	20.9	0.02
Thoracic limb bones	233 ^a	309 ^b	259 ^c	5.6	<0.01
Pelvic limb bones	430	510	423	19.0	0.08
Proportion of total bone weight, %					
Axial skeleton	48.6	48.2	45.2	2.50	0.63
Appendicular skeleton	51.3	51.7	54.8	2.50	0.63
Thoracic limb bones	18.1	19.5	20.8	0.80	0.20
Pelvic limb bones	33.3	32.2	34.0	1.91	0.82

a,b,c Means not followed by the same letter are significantly different at the 5% level of probability.

The costae were a higher percentage of total bone ($P=0.04$) in the growing animals than in either Treatments I or III (Table II.10). There was no significant difference in the percentage of carpals between the growing sheep and the load-carrying sheep (0.8 vs. 0.9%); however, the percentage of carpals in the load-carrying sheep was significantly larger (0.9 vs. 0.7%) than in the maintenance sheep.

Load-carrying sheep had total fat weights similar to the maintenance sheep and both groups had less fat ($P<0.01$) than the growing sheep (Table II.11). In the growing animals, the subcutaneous fat depot tended to be larger and the intermuscular fat depot was significantly ($P=0.04$) smaller as a proportion of total fat when compared to respective fat depots in both the maintenance and load-carrying sheep. Perirenal plus channel fat remained a relatively constant proportion of total fat (Figure II.3a). The maintenance and load-carrying sheep had nearly identical absolute weights in the three fat depots (Figure II.3b). The intermuscular and perirenal plus channel fat depots in the growing sheep were approximately three times larger whereas the subcutaneous depot was six times larger than respective depots in the load-carrying and maintenance sheep.

Similar fiber type distributions were found in the *longissimus* of load-carrying and growing sheep. Both groups were not significantly different from the maintenance sheep. The proportions of alkaline ATPase slow fibers were 12.2% in the maintenance sheep, 6.4% in the growing sheep, and 8.0% in the load-carrying sheep (Table II.12).

No differences in activity patterns were found among the three treatment groups.

D. DISCUSSION

Static load-carrying as an attempt to increase the apparent live weight of an animal has been studied previously. In most cases, load-carrying was utilized to test the effect of increased apparent live weight on bone growth and development (Tulloh and Romberg, 1963; Martin *et al.*, 1981) or on the incidence of leg weakness (Cook *et al.*, 1984; Brennan, 1986). Skjervold *et al.* (1963) and Saville and Smith (1966) were the only researchers who increased the apparent live weight borne by the hind limbs in an attempt to affect muscle growth and

Table II.10 Bone distribution (individual bone weights as a percentage of half carcass' bone weight) of maintenance, growing and load-carrying sheep.

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Number of animals	2	2	2	—	—
Half carcass bone weight, kg	1.31	1.58	1.26	0.08	0.12
Cervical vertebrae	8.9	8.9	7.4	1.58	0.76
Thoracic vertebrae	13.1	11.3	11.8	1.02	0.54
Lumbar vertebrae	7.9	6.5	7.1	1.43	0.81
Costae	12.0 ^a	14.1 ^b	11.5 ^a	0.41	0.04
Sternum and costal cartilage	6.7	7.4	7.5	0.56	0.62
Scapula	4.6	5.0	5.2	0.15	0.14
Humerus	7.2	7.3	8.1	0.29	0.18
Radio-ulna	5.6	6.5	6.5	0.40	0.31
Carpus	0.7 ^a	0.8 ^{a,b}	0.9 ^b	0.04	0.04
Os coxae	11.1	11.0	10.0	0.63	0.46
Femur	9.9	9.4	10.6	0.52	0.36
Tibia	8.3	7.8	8.7	0.60	0.66
Patella	0.7	0.7	1.0	0.09	0.15
Tarsus	3.3	3.3	3.6	0.29	0.62

^{a,b} Means not followed by the same letter are significantly different at the 5% level of probability.

Table II. 11 Fat partitioning (fat depot weight as a percentage of half carcass fat weight) of maintenance, growing and load-carrying sheep.

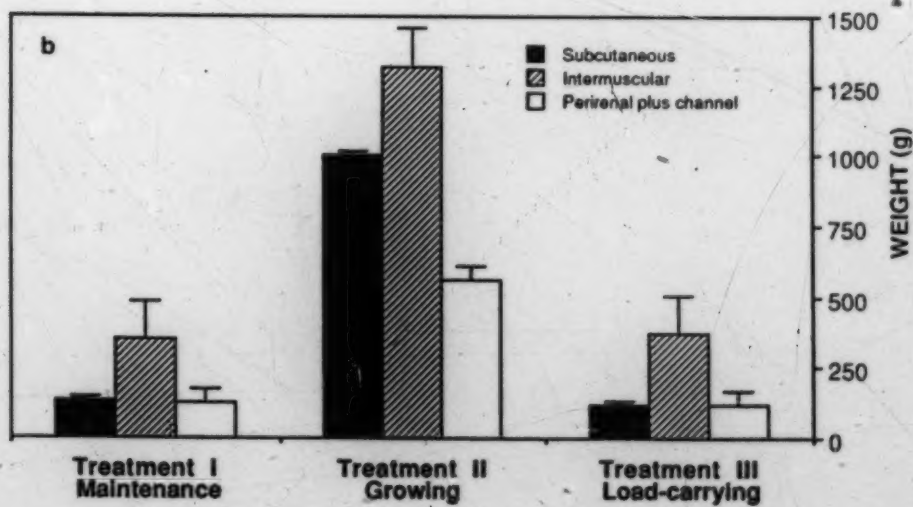
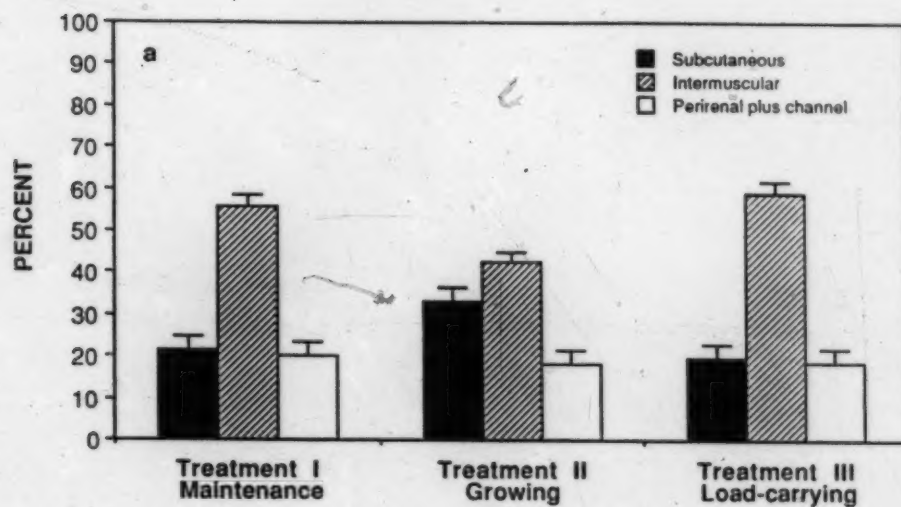
	Treatment I Maintenance	Treatment II ^a Growing	Treatment III Load-Carrying	S.E.M.	Probability
Number of animals	2	2	2	—	—
Half carcass fat weight, kg	0.62 ^a	3.09 ^b	0.63 ^a	0.23	<0.01
Subcutaneous fat	21.6	33.0	19.8	3.32	0.12
Intermuscular fat	55.8 ^a	42.5 ^b	59.1 ^a	2.54	0.04
Perirenal plus channel fat	20.3	18.1	18.8	3.20	0.89

^{a, b} Means not followed by the same letter are significantly different at the 5% level of probability.

Table II. 12 Percentage 'fast' and 'slow' fiber types in samples from the Longissimus muscle of maintenance, growing and load-carrying sheep.

	Treatment I Maintenance	Treatment II Growing	Treatment III Load-Carrying	S.E.M.	Probability
Number of animals	2	2	2	—	—
Fast fibers, %	87.8	93.5	92.0	2.07	0.28
Slow fibers, %	12.2	6.5	8.0	2.07	0.28

Figure II.3 Fat distribution in maintenance, growing and load-carrying sheep: (a) as a percentage of half carcass fat weight; (b) as absolute weights



distribution.

In this experiment, the load-carrying sheep required approximately 9% more feed than the maintenance sheep to maintain body weight. The additional energy used by the load-carrying sheep would have been expended either on the development of tension in the muscles and for mechanical work (Pandolf *et al.*, 1977) or on an increased basal metabolic rate. Unfortunately, most of the other researchers who used static load-carrying were not interested in the energy cost of load-carrying so either feed intakes were not reported or maintenance and load-carrying animals were fed the same amount of feed. Morrison *et al.* (1968) and Murray *et al.* (1974a) used an endurance form of exercise in pigs and showed that the energy cost of exercise is relatively low.

The liver was the only organ which appeared to increase as a percentage of carcass weight in the load-carrying sheep. This is probably due to the slightly higher feed consumption in the load-carrying animals since it has been shown that liver weight can be affected by the amount of feed consumed (Murray and Slezacek, 1980; Price and White, 1985). Hatai (1915) investigated changes in organ weight in exercised albino rats and reported the changes as differences between deviations from predicted values at similar body lengths of the exercised and maintenance rats. Hatai (1915) found an increase in the weight of the heart, kidneys, liver and testes and a decrease in the weight of the spleen in exercised rats. Even when the observed values were calculated as a percentage of body weight, the proportion of heart, kidneys, liver and testes remained higher in the exercised rats. However, there was no decrease in the proportion of spleen in the exercised rats.

There were no significant changes in dissectible muscle, fat or bone in the load-carrying sheep in this experiment. These results support those of Bull and Rusk (1942) who found no difference in dissected lean, fat and bone in exercised steers. However, Skjervold *et al.* (1963) reported a significant increase in the amount of fat and no increase in the weight of meat in the hams of load-carrying pigs. Martin *et al.* (1981) found a significant reduction in skinfold thickness (purportedly a measure of fatness, but also affected by the tonus of the underlying muscle) in load-carrying beagles. Peo *et al.* (1970) reported a

significant difference in the percentage of lean cuts as a result of exercise in pigs but omitted to report the direction of the change.

The percentage of connective tissue was greater in the load-carrying sheep, due mainly to the presence of a greater proportion of *ligamentum nuchae*. Kiiskinen (1977) found a transient increase in the dry weight of the Achilles tendon in exercised mice. Tipton *et al.* (1975) found a significant increase in ligament weight in exercised rats; this increase corresponded to an increase in the amount of collagen per ligament rather than an increase in collagen per unit of dry weight. It is possible that the increased load, especially over the thoracic vertebrae, may have resulted in an increase in the weight of the *ligamentum nuchae*.

There are few studies with which to compare our muscle data since few researchers have carried out full dissections of individual muscles. Despite this, numerous reports in the literature suggest that individual muscle weight does increase in response to weight lifting (Gonyea and Bonde-Petersen, 1978), endurance exercise (Saville and Whyte, 1969), compensatory hypertrophy (Gutman and Hajek, 1971; Turto *et al.*, 1974; Gollnick *et al.*, 1981) and stretch induced hypertrophy (Ashmore and Summers, 1981; Sparrow, 1982; Kane *et al.*, 1984). If the weight of an individual muscle increases, then either total muscle mass must increase or the distribution of muscle must change.

Load-carrying in this experiment did not result in an increase in total muscle mass. These results support those of Murray *et al.* (1974a) who found no increase in total muscle mass in exercised pigs. Butterfield (unpublished, cited in Berg and Butterfield, 1976) also found no increase in total muscle mass in calves exercised by jumping hurdles when compared to inactive controls.

However, some changes in muscle distribution did occur in this experiment. Muscle weight shifted from the neck (Group 9) to the proximal thoracic limb (Group 5). Muscles in Group 5 function mainly in extension, flexion and abduction of the shoulder joint and the elbow joint (May, 1964). The *supraspinatus* acts as a ligament of the shoulder joint and functions in extension of this joint. The *deltoideus* functions in flexion and abduction of the shoulder joint. The long head of the *triceps brachii* functions in extension of the elbow joint.

These muscles are all involved in maintaining joint angles and the subsequent posture of the animal (Davies, 1981). Since the load-carrying sheep tended to stand in a 'braced' position, muscle weight may have shifted to these muscles to accomodate the change in posture.

Muscles in Group 9 function mainly in support and movement of the head and neck. The *splenius* functions partly in supporting and raising the head and neck (May, 1964; Berg and Butterfield, 1976). The *longissimus capitis and atlantis* functions in extension of the head and neck and the *longus colli* functions in flexion of the neck. The decreased proportion of muscle in Group 9 may have contributed to the increase in weight of the *ligamentum nuchae* as this ligament assists the extensor muscles of the head and neck in supporting the weight of the head.

The load-carrying sheep in this experiment appeared to be able to alter bone weight distribution by preferentially laying down bone in the leg bones, particularly in the thoracic limb, to meet the need for a fortified appendicular skeleton. Mechanical stresses and strains are considered essential for normal bone growth (Anderson *et al.*, 1971). Kiiskinen and Heikkinen (1978) concluded that continuous pressure equivalent to body weight is more important than physical activity in maintaining normal calcium and nitrogen balance in bones. Tulloh and Romberg (1963) attempted to overload the long bones by artificially increasing body weight. They found that the width of the distal head of the metacarpal increased in load-carrying animals. This agrees with Wolf's law (Lakes and Saha, 1979) that bones adapt to stress in such a way as to become better able to support the stress. Other researchers looked at the effect of exercise on bone growth and found an increase in bone weight (Saville and Whyte, 1969), a conservation of bone (Anderson *et al.*, 1971), and an increase in density and a decrease in volume (Kiiskinen, 1977).

The front feet (which includes the metacarpal and phalangeal bones) were also heavier in the load-carrying sheep than in the maintenance sheep in this experiment. This lends additional support to the theory that the load-carrying sheep had fortified thoracic limb bones. Since the show blankets tended to ride forward on the sheep, placing most of the weight over the thoracic limbs, this response was not unexpected.

Load-carrying had no significant effect on either the total amount or distribution of fat in this experiment. Skjervold *et al.* (1963) found an increase in caul fat in pigs forced to feed from elevated feeders. Other researchers looking at the effect of treadmill endurance exercise in farm animals found an increase in dissectible fat or backfat depth with either no difference in feed intake in pigs (Mandigo *et al.*, 1971; Murray *et al.*, 1974a) or lower feed intake in sheep (Spaeth *et al.*, 1967). These results contrast sharply with similar experiments on the effect of treadmill exercise in rats where fat content was analysed chemically (Crews *et al.*, 1969) and in man where fat content was estimated from skinfold thickness and specific gravity (Fox and Mathews, 1981). These researchers generally reported a decrease in body fat, although Fox and Mathews (1981) stated that decreases in body fat were more apparent in obese individuals than in lean individuals. The observed changes in fat content in exercised farm animals may be due to changes in fat distribution to facilitate thermoregulation.

It appears that the load-carrying sheep, although unable to change their proportion of muscle:fat:bone, were able to redistribute the weight of these tissues to support the additional load. Bones in the front leg increased in weight in response to the compressional forces of load-carrying (Bassett and Becker, 1962; Malina, 1969). This is thought to occur due to piezo-electric properties which convert stress to electrical stimuli which then affect neighbouring osteogenic tissue (Bassett and Becker, 1962; Lakes and Saha, 1979). Tensile forces exerted by the muscles in maintaining limb posture may also have caused an increase in bone weight but, in general, tensile forces are thought to be less important than compressional forces (Malina, 1969). The increase in tension in the limb posture-maintaining muscles was probably responsible for the shift in muscle weight to the muscles of the proximal thoracic limb; passive tension is a known stimulator of muscle growth (Goss, 1978). The depletion of muscle weight in the neck may have resulted in an increase in the load which the *ligamentum nuchae* had to support, resulting in hypertrophy of this ligament.

E. CONCLUSION

Significant changes in muscle and bone distribution occurred in this pilot study, despite small numbers of animals. Load-carrying sheep were capable of adapting to the increased load by shifting bone weight from the axial skeleton to the appendicular skeleton and by shifting the proportion of muscle from head and neck to the proximal thoracic limb. This shift in muscle weight may have resulted in hypertrophy of the *ligamentum nuchae*.

The restricted feed intake of the load-carrying sheep necessitated shifts in tissue distribution; increases in total muscle weight and in total bone weight were selected against by the low feed intake. It is possible that animals on full feed would have increased muscle and bone mass without altering distribution. A further experiment was planned to investigate the effects of load-carrying on muscle growth and development in sheep fed *ad libitum*.

Chapter III

THE EFFECTS OF STATIC LOAD-CARRYING ON GROWTH AND DEVELOPMENT OF SHEEP FED *AD LIBITUM*

A. INTRODUCTION

In the previous experiment (Chapter II), it was found that static load-carrying did not increase the total muscle weight of load-carrying sheep. However, slight shifts in muscle distribution occurred. These shifts may have been a direct response to tensions resulting from an effort to maintain postural integrity (Gutmann *et al.*, 1971; Gonyea and Ericson, 1976; Davies, 1981) or a response to tensions exerted by increased bone growth in the thoracic limb (Goss, 1978). This does not agree with the popular weight lifting lore which claims large increases in individual muscle weights in response to overload exercise (Katch *et al.*, 1980).

Several researchers have investigated the effects of feed restriction on muscle growth and have concluded that feed restriction can cause a decrease in muscle growth, and during periods of severe restriction can even result in muscle atrophy (Butterfield and Berg, 1966a; Rowe, 1968; Price, 1977). Goldspink (1964) reported that the combined effects of feed restriction and exercise resulted in no increase in muscle weight in mice, although the muscle fibers of the exercised animals were considerably larger. Goldberg *et al.* (1975) suggested that compensatory hypertrophy of the *soleus* can be induced in fasting rats but it is not known whether this was a result of muscle redistribution (as the previous experiment suggests) or whether total muscle weight actually increased. Similarly, stretch-induced hypertrophy occurs in feed-restricted chickens; however, it is difficult to distinguish hypertrophy of the stretched muscle from atrophy of the contralateral control muscle on a muscle weight basis (Kane *et al.*, 1984).

The lack of gross hypertrophy in the previous experiment may have been due to the confounding effects of feed restriction and load-carrying on muscle growth (Goldspink and Ward, 1979). The following experiment was designed to remove one of the confounding effects by examining the effect of static load-carrying on sheep fed *ad libitum*.

B. MATERIALS AND METHODS

Twelve shorn, Suffolk-cross wethers, approximately 6 months of age, were weighed and randomly assigned to two treatments (Treatment I - control, Treatment II - load-carrying). Sheep in the load-carrying group were fitted with modified canvas show blankets as described in the previous experiment (Plate II.1). The sheep were drenched with Thibendazole and 1 ml of a Vitamin A-D-E mixture was administered subcutaneously. The animals were housed in individual pens and fed a pelleted diet consisting of 40% alfalfa and 60% barley based-concentrate, *ad libitum* (Table III.1). The sheep were allowed one week to adjust to their new environment and to the show blankets. On days 6, 16 and 42 of the trial, lead weights were added to maintain a load of about 35-40% of body weight (Figure III.1). Weight gain and feed consumption were recorded daily. Animals were slaughtered when their live weight reached approximately 52 kg.

The slaughter and dressing procedure (described previously in Chapter II) had the following modifications: the full alimentary tract was weighed; the front feet were disarticulated distally to the metacarpals and the hind feet were disarticulated distally to the metatarsals. The carcasses were weighed and split down the backbone. The metacarpal bone was dissected from the right front leg and the following traits were measured: length, weight, circumference (at its minimum along the shaft of the bone), maximum width of the proximal and distal heads, and width of the proximal condyles. Shape ratios (length:proximal head width; length:distal head width; length:proximal condyle width) were calculated. In addition, metacarpal shaft volume was calculated ($\text{length} \times \text{circumference}^2 + 12.57$).

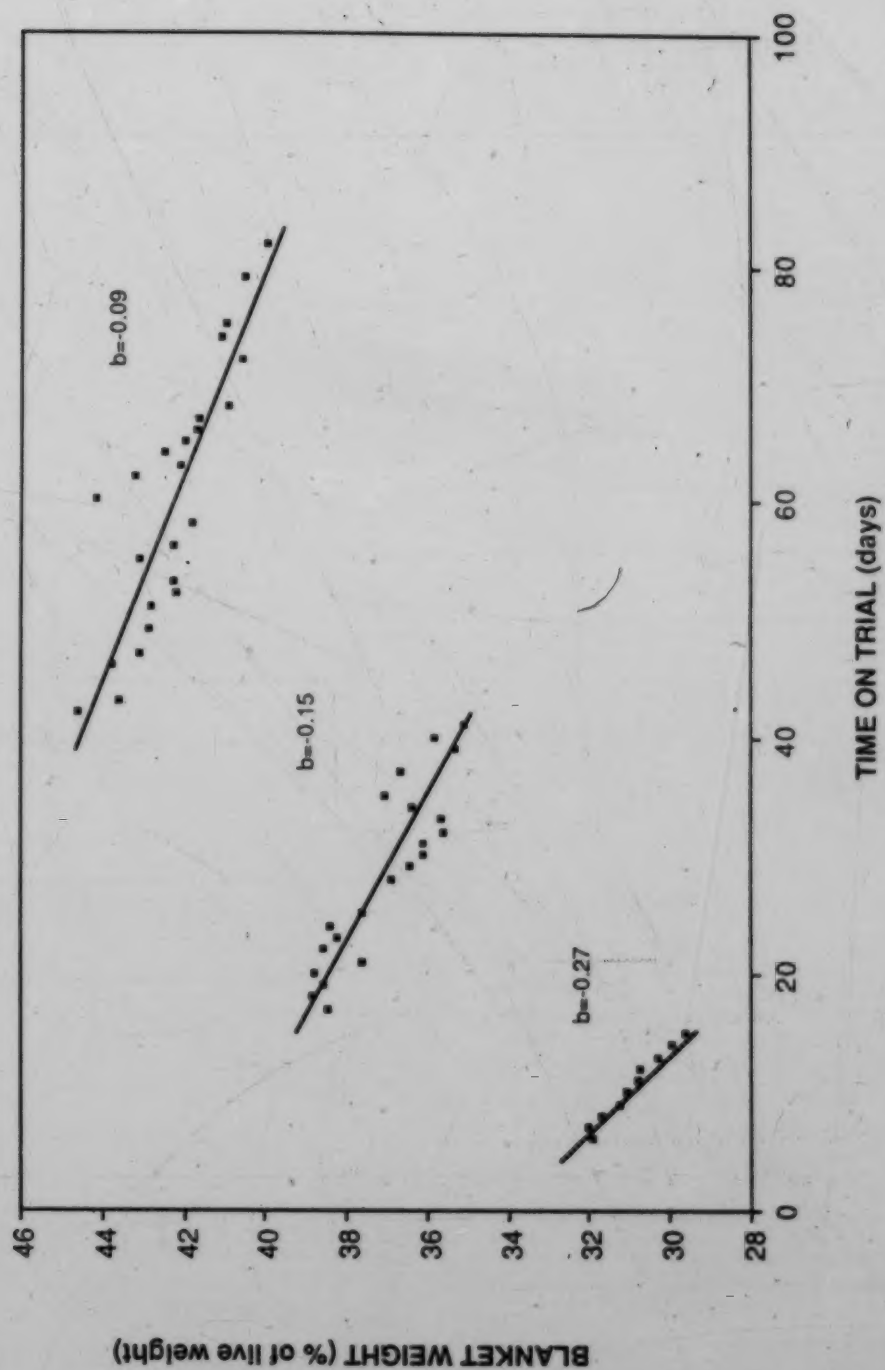
The left side of the carcass was chilled for 48 hours at 6° C. The chilled left side was then anatomically dissected and individual muscles and bones were weighed (Butterfield and May, 1966). Half carcass fat was separated into three depots (subcutaneous, intermuscular, and perirenal plus channel fat) and weighed. Connective tissue (including tendons severed at the point where muscle tissue began, small pieces of cartilage removed from the joints, and pieces of fascia) was weighed. The weight of the *ligamentum nuchae* was recorded separately.

Table III.1 The formulation of the pelleted diet which was fed to control and load-carrying sheep *ad libitum*.

	Composition (g.kg^{-1}) [†]	Analysis (dry matter basis, g.kg^{-1})
Alfalfa pellets	400	Crude protein 166
Barley	334	Calcium 12.2
Oats	67	Phosphorus 3.2
Soybean meal	100	
Alfalfa meal	67	Digestible energy, MJ.kg^{-1} 12.46
Molasses	16	

[†] To 100 kg of this diet were added 800 g salt, 800 g ground limestone and 33 g Vitamin A-D-E (10,000,000 i.u./kg Vit A, 1,650,000 i.u./kg Vit D, 10,000 i.u./kg Vit E).

Figure III.1 Blanket weight (expressed as a percentage of body weight) with time on trial



The dissected left side was then ground, mixed, reground, mixed, subsampled and analysed for dry matter, crude protein, crude fat, and ash (AOAC, 1980).

Weights of individual muscles, bones, fat depots, and offal components were expressed as absolute values and as proportions of the appropriate carcass tissue weight. Total muscle, bone and fat depot weights were expressed as absolute values and as proportions of carcass weight. All data were analysed using a one-way analysis of variance to compare treatments. Average daily gains were calculated using a regression analysis of live weight with time on trial. SPSSX (1983) was used for statistical analyses.

C. RESULTS

The initial weights of the two groups were similar by design (Table III.2). The final weight of the control group was significantly higher than that of the load-carrying group (56.1 vs. 48.8 kg) despite both groups having been fed *ad libitum*. The actual pattern of live weight gain with time on trial is shown in Figure III.2. There was a significant difference ($P < 0.01$) in average daily gain between the load-carrying and control treatments (0.22 vs. 0.35 kg.day⁻¹). The artificial average daily gain in the load-carrying sheep (loaded) was not significantly different from that of the control sheep (0.41 vs. 0.35 kg.day⁻¹). Control sheep ate 5% more feed in a shorter time period (76.8 vs. 91.8 days) than the load-carrying sheep. Load-carrying sheep required 3.5 kg more feed than control sheep per kg of gain (9.9 vs. 6.4 kg feed.kg gain⁻¹).

The empty body weight (live weight minus the full alimentary tract weight) was significantly greater in the control sheep than in the load-carrying sheep (44.8 vs. 39.8 kg; Table III.3). The proportion of front feet in the load-carrying sheep was significantly ($P < 0.01$) larger than in the controls (0.55 vs. 0.49%). The proportions of alimentary tract and liver were significantly smaller in the load-carrying sheep than in the control sheep. The spleen, bladder and penis tended to be a greater proportion in the load-carrying sheep than in the control sheep ($P = 0.07$, $P = 0.01$, $P = 0.09$, respectively).

Table III.2 Live animal performance of control and load-carrying sheep.

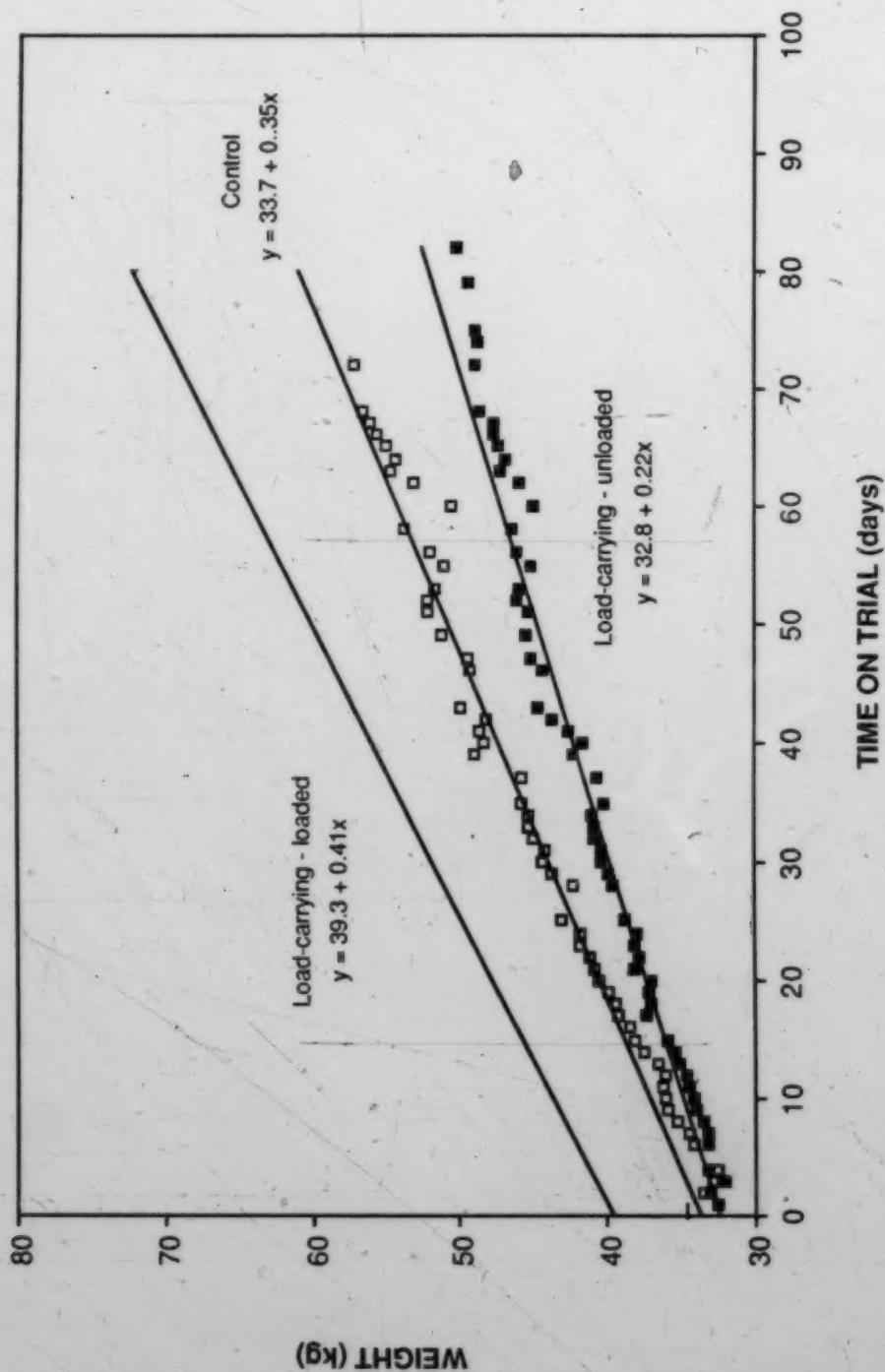
	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Number of animals	6	6	—	—
Initial weight, kg	32.7	34.0	1.3	—
Final weight, kg	56.1	48.8	1.3	<0.01
Weight gain, kg	23.4	14.8	1.6	<0.01
Average daily gain, kg.day ⁻¹	0.35	0.22	0.02	<0.01
Feed consumption, kg	147.3	140.0	3.3	0.14
Feed conversion efficiency, kg.kg ⁻¹	6.4	9.9	0.6	<0.01
Average no. days on trial	76.8	91.8	2.2	<0.01

Table III.3 Offal components (as a percentage of empty body weight[†]) of control and load-carrying sheep.

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Number of animals	6	6	—	—
Empty body weight [†] , (kg)	44.8	39.8	1.03	<0.01
Fleece	14.4	14.9	0.55	0.55
Head	4.3	4.6	0.12	0.12
Front feet	0.49	0.55	0.01	<0.01
Hind feet	0.51	0.50	0.02	0.59
Alimentary tract	20.1	18.4	0.51	0.04
Heart	0.57	0.48	0.06	0.31
Lungs	2.3	1.9	0.14	0.13
Liver	2.5	1.8	0.16	0.01
Kidneys	0.32	0.31	0.02	0.83
Spleen	0.18	0.27	0.03	0.09
Bladder	0.03	0.06	<0.01	0.01
Penis	0.11	0.15	0.01	0.07

[†] Live weight minus the full alimentary tract.

Figure III.2 Increases in live weight of control and load-carrying sheep with time on trial



Load-carrying sheep had a higher dressing percentage (carcass weight as a percentage of live weight) than in the control sheep (56.9 vs. 53.2%; Table III.4). Even when the carcass was expressed as a percentage of live weight minus the alimentary tract, the dressing percentage was still higher in the load-carrying sheep than in the controls. Load-carrying sheep had a significantly higher percentage of muscle and bone and a lower percentage of fat than control sheep ($P=0.03$, $P=0.01$, $P=0.01$, respectively). The muscle:bone ratio tended to be larger in the control sheep than in the load-carrying sheep (4.7 vs. 4.3).

There were no significant differences in chemically determined water, protein or ash content between sheep of the two treatments (Table III.5). However, there was a tendency for the percentage of chemically determined fat to be lower in the load-carrying sheep than in the controls ($P=0.09$).

Control and load-carrying sheep were similar for all anatomical muscle groups (Table III.6), except for the muscles in the abdominal wall (Group 4). The proportion of muscle in Group 4 was significantly larger in the load-carrying sheep than in the control sheep (10.4 vs. 12.0%; $P<0.01$).

Few muscle weights were substantially different as proportions of carcass muscle weight between the two groups of sheep. However, the following muscles were significantly smaller in the load-carrying sheep than in the control sheep when expressed as a percentage of half carcass muscle weight (Table III.7): *gluteus accessorius*, *rectus femoris*, *anconaeus* ($0.10 \geq P > 0.05$); *spinalis dorsi* ($0.05 \geq P > 0.01$); *vastus intermedius*, *triceps brachii* (*caput longum*), *flexor digitorum sublimis* ($P \leq 0.01$). The following muscles were a significantly larger proportion in the load-carrying sheep than in the control sheep: *sartorius*, *articularis genu*, *flexor digitorum profundus* ($0.10 \geq P > 0.05$); *peroneus longus*, *quadratus lumborum*, *obliquus externus abdominis*, *obliquus internus abdominis*, *deltoideus*, *obliquus capitis caudalis* ($0.05 \geq P > 0.01$); *extensor digiti quarti proprius*, *transversus abdominis*, *diaphragm* ($P \leq 0.01$).

Total bone weight did not differ between control and load-carrying sheep (1.69 vs. 1.78 kg; Table III.8). Load-carrying sheep had a slightly lower proportion of bone weight in the axial skeleton (45.7 vs. 46.2). Load-carrying sheep had slightly lower proportions of atlas

Table III. 4 Half carcass dissection of control and load-carrying sheep.

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Number of animals	6	6	—	—
Half carcass weight, kg	14.4	13.2	0.3	0.02
Dressing percentage [†]	53.2	56.9	1.0	0.03
Carcass percentage ^{††}	66.5	69.6	1.0	0.05
Muscle [‡]	52.8	55.8	0.8	0.02
Fat [‡]	35.2	30.4	1.1	0.01
Bone [‡]	11.3	13.0	0.4	0.01
Tendon [‡]	0.7	0.8	0.1	0.37
Muscle:bone ratio	4.7	4.3	0.1	0.08

[†] Warm carcass weight expressed as a percentage of live weight.

^{††} Warm carcass weight expressed as a percentage of live weight minus full alimentary tract weight.

[‡] Expressed as a percentage of half carcass weight.

Table III.5 Chemical analysis of the ground left side of control and load-carrying sheep.

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Number of animals	6	6	-	-
Water, %	50.4	51.7	0.8	0.31
Protein, %	13.2	14.0	0.4	0.23
Fat, %	32.8	30.6	0.8	0.09
Ash, %	3.5	4.0	0.3	0.23

Table III.6 Anatomical muscle group weights (as a percentage of half carcass muscle weight) of control and load-carrying sheep.

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Number of animals	6	6	—	—
Half carcass muscle weight, kg	7.60	7.36	0.23	0.44
Muscle group				
1. Proximal pelvic limb	29.8	29.2	0.31	0.15
2. Distal pelvic limb	4.5	4.6	0.14	0.59
3. Around spinal column	16.4	15.8	0.40	0.31
4. Abdominal wall	10.4	12.0	0.28	<0.01
5. Proximal thoracic limb	13.5	13.1	0.25	0.18
6. Distal thoracic limb	3.2	3.1	0.09	0.43
7. Thorax to thoracic limb	8.1	8.3	0.31	0.73
8. Neck to thoracic limb	4.6	4.5	0.21	0.64
9. Neck and thorax	9.4	9.5	0.18	0.89

Table III.7 Muscle distribution (individual muscle weights as a percentage of half carcass muscle weight) of control and load-carrying sheep.

	Treatment I		Treatment II		S.E.M.	Probability
	Control		Load-Carrying			
Number of animals	6		6			
Half carcass muscle weight, kg	7.60		7.36		±0.23	0.44
Muscle Group 1 - Proximal Pelvic Limb						
1. Tensor fascia latae	1.00		1.02		±0.032	0.67
2. Biceps femoris	5.40		5.23		±0.081	0.17
3. Gluteus medius	3.32		3.55		±0.108	0.16
4. Vastus lateralis	2.39		2.47		±0.034	0.14
5. Gluteus accessorius	0.64		0.55		±0.034	0.08
6. Gluteus profundus	0.33		0.30		±0.020	0.28
7. Rectus femoris	2.29		2.16		±0.044	0.06
8. Semitendinosus	1.92		1.87		±0.083	0.65
9. Gracilis	1.00		1.00		±0.031	0.98
10. Semimembranosus	5.25		4.98		±0.107	0.11
11. Adductor femoris	2.20		2.07		±0.058	0.14
12. Pectineus	0.53		0.56		±0.013	0.13
13. Sartorius	0.19		0.23		±0.014	0.07
14. Gemellus	0.10		0.09		±0.013	0.80
15. Quadratus femoris	0.08		0.09		±0.009	0.61
16. Obturator internus et externus	0.59		0.56		±0.016	0.19
17. Vastus medialis	0.89		0.79		±0.081	0.39
18. Vastus intermedius	0.87		0.76		±0.016	0.01
19. Articularis genu	0.13		0.19		±0.021	0.06
20. Iliacus	0.71		0.74		±0.030	0.53

Continued

Table III.7 (Cont'd.)

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Muscle Group 2 - Distal Pelvic Limb				
1. <i>Gastrocnemius et soleus</i>	1.63	1.54	±0.045	0.18
2. <i>Flexor digitorum superficialis</i>	0.56	0.56	±0.013	0.13
3. <i>Extensor group</i>	0.63	0.61	±0.016	0.35
4. <i>Peroneus longus</i>	0.18	0.22	±0.010	0.03
5. <i>Extensor digiti quarti proprius</i>	0.20	0.27	±0.012	<0.01
6. <i>Tibialis anterior</i>	0.23	0.19	±0.015	0.15
7. <i>Tibialis posterior</i>	0.16	0.25	±0.039	0.16
8. <i>Popliteus</i>	0.28	0.28	±0.013	0.89
9. <i>Flexor digitorum longus</i>	0.16	0.17	±0.010	0.77
10. <i>Flexor hallucis longus</i>	0.49	0.48	±0.055	0.87
Muscle Group 3 - Spinal				
1. <i>Psoas minor</i>	0.53	0.57	±0.027	0.36
2. <i>Psoas major</i>	1.62	1.65	±0.033	0.52
3. <i>Quadratus lumborum</i>	0.30	0.37	±0.017	0.02
4. <i>Iliocostalis</i>	0.43	0.45	±0.038	0.78
5. <i>Longissimus</i>	10.21	9.84	±0.266	0.35
6. <i>Spinalis dorsi</i>	1.51	1.29	±0.070	0.05
7. <i>Multifidi dorsi</i>	1.52	1.43	±0.055	0.25
8. <i>Longissimus cervicis</i>	0.27	0.26	±0.023	0.64

Continued

Table III.7 (Cont'd.)

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Muscle Group 4 - Abdominal				
1. Cutaneous trunci	2.16	2.17	±0.105	0.95
2. Serratus dorsalis caudalis	0.24	0.23	±0.022	0.61
3. Obliquus externus abdominis	1.91	2.17	±0.079	0.04
4. Retractor costae	0.15	0.12	±0.035	0.64
5. Obliquus internus abdominis	1.27	1.69	±0.124	0.04
6. Transversus abdominis	1.37	1.88	±0.084	<0.01
7. Rectus abdominis	2.44	2.37	±0.190	0.80
8. Diaphragm	0.83	1.33	±0.071	<0.01
Muscle Group 5 - Proximal Thoracic Limb				
1. Deltoides	0.51	0.57	±0.018	0.03
2. Infraspinatus	2.17	2.07	±0.049	0.18
3. Triceps brachii (caput laterale)	0.92	0.84	±0.034	0.11
4. Teres minor	0.21	0.20	±0.015	0.75
5. Triceps brachii (caput longum)	2.80	2.50	±0.068	0.01
6. Tensor fasciae antibrachii	0.35	0.53	±0.156	0.43
7. Supraspinatus	2.24	2.09	±0.075	0.19
8. Biceps brachii	0.58	0.58	±0.011	0.71
9. Teres major	0.58	0.59	±0.019	0.73
10. Coracobrachialis	0.20	0.25	±0.020	0.12
11. Subscapularis	1.13	1.07	±0.034	0.27
12. Brachialis	0.34	0.37	±0.014	0.19
13. Brachiocephalicus	1.23	1.16	±0.084	0.59
14. Triceps brachii (caput medialis)	0.26	0.22	±0.012	0.26

Continued

Table III.7 (Cont'd.)

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Muscle Group 6 - Distal Thoracic Limb				
1. Extensor carpi radialis	0.71	0.67	±0.025	0.29
2. Extensor digitorum longus	0.17	0.17	±0.009	0.92
3. Extensor digitorum communis	0.13	0.14	±0.015	0.87
4. Extensor digitorum lateralis	0.19	0.19	±0.043	0.96
5. Extensor carpi ulnaris lateralis	0.36	0.37	±0.051	0.97
6. Abductor pollicis longus	0.07	0.06	±0.009	0.73
7. Flexor carpi radialis	0.14	0.17	±0.013	0.14
8. Flexor carpi ulnaris	0.25	0.30	±0.024	0.20
9. Flexor digitorum sublimis	0.49	0.34	±0.031	0.01
10. Flexor digitorum profundus	0.51	0.54	±0.046	0.07
11. Anconaeus	0.24	0.21	±0.007	0.07
Muscle Group 7 - Thorax to Thoracic Limb				
1. Latissimus dorsi	1.65	1.77	±0.052	0.33
2. Trapezius thoracic	0.56	0.51	±0.023	0.20
3. Serratus ventralis thoracis	1.75	1.85	±0.093	0.46
4. Pectoralis profundus	3.32	3.29	±0.077	0.76
5. Pectoralis superficialis	0.65	0.84	±0.150	0.39

Continued

Table III.7 (Cont'd.)

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Muscle Group 8 - Neck to Thoracic Limb				
1. <i>Trapezius cervicalis</i>	0.35	0.40	±0.018	0.13
2. <i>Omotransversarius</i>	0.48	0.62	±0.068	0.17
3. <i>Rhomboideus</i>	0.78	0.76	±0.036	0.66
4. <i>Serratus ventralis cervicis</i>	2.98	2.69	±0.131	0.15
Muscle Group 9 - Neck and Thorax				
1. <i>Intercostales</i>	2.78	2.47	±0.125	0.11
2. <i>Serratus dorsalis cranialis</i>	0.08	0.10	±0.029	0.62
3. <i>Scalenus dorsalis</i>	0.18	0.19	±0.046	0.86
4. <i>Cervicothoraceus</i>	0.06	0.11	±0.039	0.44
5. <i>Splenius</i>	0.22	0.23	±0.014	0.71
6. <i>Scalenus ventralis</i>	0.54	0.53	±0.026	0.65
7. <i>Longus capitis</i>	0.23	0.24	±0.012	0.33
8. <i>Intertransversarius longus</i>	0.29	0.31	±0.043	0.70
9. <i>Longissimus capitis and atlantis</i>	0.34	0.32	±0.030	0.93
10. <i>Intertransversarii cervicis</i>	0.53	0.61	±0.053	0.34
11. <i>Semispinalis capitis</i>	1.05	1.05	±0.041	0.96
12. <i>Obliquus capitis caudalis</i>	0.59	0.80	±0.058	0.03
13. <i>Rectus thoracis</i>	0.07	0.11	±0.030	0.39
14. <i>Transversus thoracis</i>	0.29	0.25	±0.022	0.33
15. <i>Longus colli</i>	0.68	0.79	±0.074	0.32
16. <i>Multifidi cervicis</i>	0.60	0.55	±0.047	0.54
17. <i>Sternoccephalicus</i>	0.82	0.71	±0.139	0.57

Table III.8 Bone distribution (individual bone weights as a percentage of half carcass bone weight) of control and load-carrying sheep.

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Number of animals	6	6		
Half carcass bone weight, kg	1.69	1.78	0.07	0.39
Axial skeleton, %				
Atlas	146.2	45.7	0.46	0.46
Cervical vertebrae	1.7	1.4	0.10	0.04
Thoracic vertebrae	8.0	7.6	1.28	0.83
Lumbar vertebrae	9.5	8.2	0.48	0.08
Costae	5.8	9.0	0.62	<0.01
	13.7	12.5	0.46	0.10
Sternum and costal cartilage	7.3	6.9	0.34	0.41
Appendicular skeleton, %				
Scapula	53.8	54.3	0.46	0.46
Humerus	4.9	4.6	0.11	0.05
Radio-ulna	7.5	7.1	0.09	<0.01
Carpus	6.0	5.9	0.12	0.35
Metacarpal	1.0	0.9	0.08	0.41
Os Coxae	3.1	3.1	0.08	0.86
Femur	10.7	12.3	0.57	0.07
Tibia	9.4	9.4	0.19	0.90
Patella	7.9	7.8	0.19	0.92
Tarsus	0.6	0.6	0.03	0.92
	2.9	2.7	0.13	0.60

bone ($P=0.04$) and thoracic vertebrae ($P=0.08$) and higher proportions of lumbar vertebrae ($P<0.01$) and os coxae ($P=0.07$). The control sheep had a significantly higher percentage of scapula and humerus ($P=0.05$, $P<0.01$, respectively).

Linear dimensions of the metacarpal bone did not differ significantly between the control and exercised sheep (Table III.9). However, the shape ratios of length to proximal head width, and length to proximal condyle width, tended to be smaller in the load-carrying sheep ($P=0.07$, $P=0.10$, respectively). The calculated shaft volume did not differ between sheep of the two treatments.

Total fat weight of the half carcass was significantly heavier in the control sheep than in the load-carrying sheep (5.69 vs. 4.40, $P=0.01$; Table III.10). On an absolute weight basis, the load-carrying sheep had significantly less subcutaneous and perirenal plus channel fat than the control sheep ($P<0.01$, $P=0.02$, respectively). There were no significant differences between the two groups when individual fat depots were expressed as a percentage of half carcass fat weight.

D. DISCUSSION

Despite allowing the sheep to feed *ad libitum*, the load-carrying sheep consumed less feed and grew at a much slower rate than control sheep. This agrees with the results of Spaeth *et al.* (1967) who reported lower feed consumption in exercised lambs and Morrison *et al.* (1968) who reported slower rates of gain in exercised pigs. Crews *et al.* (1969) attributed the lower weight gain in exercised rats to a decrease in appetite and an increase in energy expenditure for exercise. Crews *et al.* (1969) also suggested that the degree of appetite suppression was due to the amount of stress associated with the exercise rather than the exercise *per se*.

The low growth rates in the load-carrying sheep presented a dilemma since the distribution of tissues can be influenced by live weight and stage of maturity (Lohse *et al.*, 1971; Butterfield *et al.*, 1983a,b,c; Butterfield and Thompson, 1983). To attain live weights similar to control sheep at slaughter, the load-carrying sheep would have required extra time

Table III.9 Linear dimensions and shape data from the metacarpal bone of control and load-carrying sheep.

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Number of animals	6	6	-	-
Weight, g	52.5	54.9	1.55	0.29
Length, cm	12.5	12.1	0.16	0.17
Width, cm				
proximal head	3.4	3.5	0.06	0.49
distal head	3.2	3.2	0.07	0.68
proximal condyles	3.2	3.2	0.06	0.80
Circumference, cm	5.4	5.5	0.12	0.61
Shape ratio, length:width				
proximal head	3.6	3.5	0.06	0.07
distal head	3.5	3.8	0.07	0.17
proximal condyles	3.9	3.8	0.05	0.10
Radius, cm ^a	0.85	0.87	0.02	0.61
Volume, cm ^{3b}	28.7	28.9	1.36	0.92

^aEstimated radius (radius=circumference/6.28).^bEstimated volume (volume=length x 3.14r²).

Table III. 10 Fat partitioning (absolute fat depot weights and fat depot weights as a percentage of half-carcass fat weight) of control and load-carrying sheep.

	Treatment I Control	Treatment II Load-Carrying	S.E.M.	Probability
Number of animals	6	6	—	—
Half carcass fat weight, kg	5.69	4.40	0.17	<0.01
Absolute weight basis, kg				
Subcutaneous fat	2.67	1.92	0.10	<0.01
Intermuscular fat	1.65	1.42	0.10	0.24
Perirenal plus channel fat	1.37	1.06	0.08	0.02
Proportion of half carcass fat, %				
Subcutaneous fat	47.0	43.8	1.77	0.23
Intermuscular fat	28.9	32.0	1.69	0.21
Perirenal plus channel fat	24.2	24.2	1.57	0.99

on trial. As well, as time on trial progressed, the growth rates of load-carrying sheep decreased. Rather than compare load-carrying sheep with control sheep at potentially large differences in chronological age, load-carrying sheep were slaughtered at a lighter live weight; I believed that many of the differences in body composition which had previously been attributed to exercise might have been caused by differences in slaughter weight.

Exercise has been claimed to increase lean and decrease fat content (Spaeth *et al.*, 1967; Crews *et al.*, 1969); lighter carcasses also have a higher proportion of muscle and bone and a lower proportion of fat (Berg and Butterfield, 1976; Butterfield *et al.*, 1984). Lighter carcass weights (8%) in the load-carrying sheep probably had more influence on the proportions of muscle, fat and bone than the load-carrying exercise. The chemical composition data also support these changes in muscle, fat and bone. Load-carrying sheep had a slightly larger proportion of protein and ash, and a smaller proportion of fat. Normally as carcass weight increases, the percentage of water, protein and ash decreases and the percentage of fat increases (Haeker, 1920; as cited in Berg and Butterfield, 1976).

Unfortunately, the difference in live weight at slaughter did not account for all the differences in carcass composition between the load-carrying and control sheep; load-carrying sheep appeared to be at a later stage of maturity at a lighter weight than control sheep. There were several pieces of evidence supporting this conclusion: (a) growth rates; (b) anticipated mature weights; (c) proportions of offal; (d) dressing percentage; (e) total bone weight and bone weight distribution; (f) total fat weight and fat weight distribution; and (g) muscle weight distribution. Each of these will be considered.

(a) Experimental manipulation of growth rates can result in differences in body composition at similar live weights (Murray *et al.*, 1974b; Murray and Slezacek, 1975; Murray and Slezacek, 1980); slow growing animals appeared to be at a later stage of maturity than fast growing animals. The lower growth rates in the load-carrying sheep may have resulted in both a lighter weight and a later stage of maturity at slaughter. This would have major implications to most exercise studies where all changes in composition are attributed to exercise.

(b) Not only did the load-carrying sheep grow at a slower rate but it appeared from the shape of their growth curve that they may have reached a lighter mature body weight if they had been allowed to reach maturity. Although not studied by Murray and co-workers (1974b, 1975, 1980), I assume that their slower-growing animals would have also reached a lighter mature weight had they been kept on treatment until maturity. Several authors have compared light mature weight animals to heavy mature weight animals, within a breed, at the same live weight (Jones *et al.*, 1978; Thompson *et al.*, 1979a,b; Taylor *et al.*, 1980; Butterfield and Thompson, 1983; Butterfield *et al.*, 1983a,b,c). Many of the differences in tissue distribution which they observed are similar to differences between load-carrying and control sheep.

(c) The differences in proportion of offal components between load-carrying and control sheep closely parallel differences at the same live weight between small mature size and large mature size rams. Most body organs are early maturing (Kirton *et al.*, 1972; Doornenbal and Tong, 1981), however Butterfield *et al.* (1983c) found that the penis plus bladder (combined) was later maturing. At similar live weights, rams of a small mature weight would have a larger proportion of penis plus bladder since they are closer to maturity than rams of a large mature weight; the penis and bladder were a larger proportion of empty body weight in the load-carrying sheep than the control sheep. In addition, Butterfield *et al.* (1983c) noted that, at the same shorn full live weight, the distal limbs were a non-significantly larger proportion in Merino rams of a small mature size. The distal parts of the fore-limbs were a significantly larger proportion of empty body weight in the load-carrying sheep.

(d) Lighter carcasses usually have lower dressing percentages (Field and Schoonover, 1967; Gaili and Nour, 1980a); load-carrying sheep had lighter carcass weights and higher dressing percentages. The load-carrying sheep had less gut fill which contributed to (Berg and Butterfield, 1976), but did not fully account for the higher dressing percentage in these animals. Murray *et al.* (1974b) found a higher dressing percentage in slow growing cattle than fast growing cattle compared at the same live weight. Thus, the higher dressing

percentage in load-carrying sheep was assumed to be due to a later stage of maturity at a similar live weight as control sheep.

(e) Total bone weight and bone weight distribution also supports a later stage of maturity in load-carrying sheep. Animals grown at a slow rate had significantly higher total bone weight than animals grown at a fast rate at the same dissected side weight (Murray *et al.*, 1974b; Atkins and Thompson, 1979). They attributed their results to bone development being related to both carcass weight and age (or stage of maturity). Total bone weight was slightly larger in the load-carrying sheep, although as a proportion of carcass weight total bone weight was significantly larger. Butterfield and Thompson (1983) worked with two strains of Merino rams which differed in mature weight, and compared them at the same stage of maturity and at the same live weight.

Butterfield and Thompson (1983) suggested that, in comparisons at the same weight, the largest differences in proportional composition between animals of different mature size would exist for those components for which the maturity coefficients deviated the most from 1.0. Since there may be breed differences in maturity coefficients, a direct comparison of the Merino coefficients cannot be made to the Suffolk rams in the present experiment. The most significant differences in bone weight distribution between load-carrying and control sheep were a larger proportion of lumbar vertebrae and a smaller proportion of humerus in the load-carrying sheep. The corresponding maturity coefficients for these bones (Butterfield and Thompson, 1983) were greater than 1.0 for the humerus and less than 1.0 for the lumbar vertebrae; rams of a small mature weight had a larger proportion of lumbar vertebrae and a smaller proportion of humerus than rams of a large mature weight when compared at similar total bone weights.

(f) A later stage of maturity in load-carrying sheep was also reflected in total fat weight and fat weight distribution. Fat is the latest maturing tissue, with a growth coefficient greater than 1.0 (Kirton *et al.*, 1972; Gaili and Nour, 1980b; Butterfield *et al.*, 1983a). In this experiment, the control sheep had significantly more total fat than the load-carrying sheep. This agrees with Butterfield and Thompson (1983) who reported that at equal live weights

large mature size rams were fatter than small mature size rams. Butterfield and Thompson (1983) also found that the individual fat depots and total fat matured at similar rates in both strains of Merino rams. Thus, when compared either at the same total fat weight or at the same stage of maturity, individual fat depots comprised similar proportions of total fat weight. There were no significant differences in the partitioning of fat expressed as a percentage of total fat between load-carrying and control sheep.

(g) The detailed muscle weight distribution data is difficult to interpret. Other studies are not closely comparable since they either used a different breed of sheep, had different growth rates or cover a different live weight range than this study (Thompson *et al.*, 1979a). Despite these difficulties, an attempt was made to relate changes in muscle weight distribution to the theme of this discussion; load-carrying sheep were at a later stage of maturity than control sheep.

The only anatomical muscle group which differed between the load-carrying and control sheep was the abdominal muscle group which was significantly heavier in the load-carrying sheep than the control sheep. This does not agree with the results of Butterfield *et al.* (1983b) who reported that muscle groups 1 and 2 (proximal and distal pelvic limb, respectively) were significantly lighter and muscle groups 4, 8, and 9 (abdominal wall, neck to thoracic limb, neck and thorax, respectively) were significantly heavier in small mature size Merino rams than in large mature size Merino rams at the same total muscle weight. It is very unlikely that the increased proportion of abdominal muscle was a result of load-carrying. In the present study, load-carrying sheep ate less feed so the increased proportion of abdominal muscle was probably not an adaptation to weight of feed in the alimentary tract, although the rate of passage of the feed could have been affected by the stress of load-carrying. These results may reflect a higher intramuscular fat content in the abdominal muscles in the load-carrying sheep (Murray and Slezacek, 1975). However, the most likely explanation is that Butterfield *et al.* (1983b) compared their rams at 8.24 kg total side muscle whereas our rams were compared at approximately 7.5 kg total side muscle which would result in a different muscle distribution.

The differences in individual muscle weight distribution between the load-carrying and control sheep can be compared using growth coefficients (Berg and Butterfield, 1976). A muscle which grows at the same rate as total muscle weight remains a constant proportion of total muscle weight and has a growth coefficient not different from 1.0. A muscle which grows at a slower rate than total muscle weight will become a decreasing proportion of total muscle weight and will have a growth coefficient less than 1.0. A muscle which grows at a faster rate than total muscle weight will become an increasing proportion of total muscle weight and will have a growth coefficient greater than 1.0.

Lohse *et al.* (1971) reported growth coefficients for a strain of Peppin Merino sheep from 3.1 to 35.9 kg live weight, hence these animals were lighter and of a different breed than the animals used in the present study. However, although some breed differences occur in muscle distribution (Seebeck and Tulloh, 1968), they are usually small. Thus, the growth coefficients reported by Lohse *et al.* (1971) were used for comparison between treatments in cases where significant differences in muscle distribution occurred between the control and load-carrying sheep.

Individual muscle weights as a proportion of total muscle mass were compared between load-carrying and control sheep at a total muscle mass of 7.36 kg and 7.60 kg, respectively. Where the proportion of an individual muscle had changed between load-carrying and control sheep in the direction indicated by the growth coefficient, the changes in distribution were assumed to be due to a normal growth response as total muscle weight varied from 7.36 to 7.60 kg. The proportion of the muscles along the spinal column and the abdomen changed in an opposite direction than indicated by their growth coefficients.

The *quadratus lumborum* and the *spinalis dorsi* of the spinal muscles (Group 3) are classified as monophasic muscles with growth coefficients close to 1.0 (Lohse *et al.*, 1971). The muscles in the abdominal group (*obliquus externus abdominis*, *obliquus internus abdominis*, *transversus abdominis* and *diaphragm*) all have high or average/high growth impetus. Therefore, as a proportion of total muscle, these muscles should be increasing as total muscle weight increases; the opposite occurred. It is possible that these muscles undergo a change in

growth impetus beyond the limits of the data used by Lohse and his coworkers. However, a more likely explanation is that individual muscle distribution was affected by stage of maturity as well as by total muscle weight.

Thus, many of the differences which occurred between load-carrying and control sheep may be attributed to a later stage of maturity in the load-carrying sheep at a lighter live weight.

Other than the reduced growth rate in load-carrying sheep, only the change in shape of the metacarpal bone can be directly attributed to the load-carrying exercise. The metacarpals of load-carrying sheep tended to have a smaller ratio of length to proximal head width. This agrees with the results of Tulloh and Romberg (1963) who reported a significantly greater width of the distal head of the metacarpal in load-carrying lambs and Kiiskinen (1977) who reported a decreased length of femoral bones in exercised mice. However, Martin *et al.* (1981) found no change in the linear dimensions of the tibia of adult beagles carrying loads of up to 130% of their body weight for 48 weeks; the lack of response was probably due to the fact that the beagles were adult dogs rather than actively growing dogs. Malina (1969) attributed the increase in bone width to muscular tensions and the decrease in bone length to growth-retarding compressive forces. In this experiment the shorter bone length may have been attributed to an earlier stage of maturity in the load-carrying sheep. However, the rest of the bone data indicated that, if anything, load-carrying sheep had a bone distribution corresponding to a later stage of maturity.

The smaller proportion of liver and alimentary tract in the load-carrying sheep than in the control sheep was probably due to their lower average daily feed intake (Murray and Slezacek, 1980; Price and White, 1985).

E. CONCLUSION

Despite carrying loads between 30 and 45% of live weight for almost 13 weeks, the only changes attributable to load-carrying were a slightly shorter and wider metacarpal bone and a decrease in average daily feed intake, resulting in a decrease in average daily gain. All

the other differences between the load-carrying and control sheep appeared to be due to different stages of maturity at similar live weights. The difference in stage of maturity may have resulted only from differences in growth rates. Other researchers (Donaldson, 1935; Malina, 1969; Kissinen, 1977) have reported that exercise tends to accelerate the growth of actively growing tissues. Accelerated growth of actively growing tissues would result in a tissue distribution similar to a later stage of maturity. This finding has major implications to most exercise studies since the effect of maturity, live weight and growth patterns of various tissues are virtually ignored and all changes, even changes in distribution, are attributed to the effect of the exercise.

The load-carrying sheep were only two weeks older than the control sheep at the time of slaughter. However, some of the differences between the load-carrying and control sheep appeared to be as large as differences reported by Butterfield *et al.* (1983a,b,c) and Butterfield and Thompson (1983) even though their rams of a small mature weight were between 10 and 15 weeks older than their rams of a large mature weight. It seems unlikely that the differences could have resulted from only a two week difference in chronological maturity corresponding to a 3.3 kg gain in live weight. Perhaps the reduced average daily gains associated with load-carrying caused an acceleration of maturity such that even at the same chronological age, load-carrying sheep were closer to their mature weight and mature tissue distribution than control sheep.

Chapter IV

THE EFFECTS OF A PROGRESSIVE-RESISTANCE-EXERCISE ON GROWTH, DEVELOPMENT AND MEAT QUALITY OF SHEEP

A. INTRODUCTION

The two static loading experiments (Chapters II and III) provided little support for the theory that total muscle weight is governed by the load it has to support. Methods used in the first experiment (Chapter II) yielded small changes in muscle distribution but no increase in total muscle weight. The second experiment resulted in changes in muscle distribution that were attributed to the load carrying sheep being closer to their mature body weight than the control sheep. However, in the first experiment, bone weight distribution was affected by the load carrying and in the second experiment the shape of the metacarpal bone was affected by the load carrying. It seemed probable that the lack of muscular hypertrophy was due to the ability of the skeleton to support much of the increased load while muscular support was only necessary during movement and to maintain postural integrity. Thus it was necessary to design a dynamic exercise which would place the musculature in a more active role.

Maximum muscular hypertrophy is thought to occur when muscle performs against workloads that are above those normally encountered (Fox and Mathews, 1981). The success of this principle has been well established in the laboratory models of exercise hypertrophy such as tenotomy (Denny Brown, 1960; Macková and Hník, 1972; Goldberg *et al.*, 1975), excision of synergistic muscles (Reitsma, 1970; Schiaffino and Pierobon Bormioli, 1973; Baldwin *et al.*, 1977) and chronic muscle stretching (Sola *et al.*, 1973; Holly *et al.*, 1980; Ashmore and Summers, 1981). In humans, these extreme models are not applicable, hence progressive-resistance-exercise (PRE) has been used to maintain an overload on the muscle (Fox and Mathews, 1981). As a muscle gains in strength and endurance it is continually challenged by a greater resistance.

In addition, human training programs are designed to produce gains in individual muscles or muscle groups which are utilized in specific activities. In farm animals, the muscle groups of importance are those located in the parts of the body yielding higher priced cuts of meat. The following experiment was designed to produce muscular hypertrophy in the hindquarter of sheep by incorporating the principles of progressive-resistance-exercise.

B. MATERIALS AND METHODS

Preliminary work

A jumping exercise was suggested as most logical to exercise the muscles in the hind limb of the sheep (Belcastro, pers. comm.). The stimulus required to have the sheep perform the exercise, and the initial levels of frequency, duration and intensity of the exercise were determined in a preliminary trial.

A positive stimulus (food) was initially offered to encourage the sheep to jump up onto a platform. However, the frequency of performing the exercise could not be controlled, even with intermittent feeding. The sheep would also have had to be kept on a sub-maintenance diet to ensure that they would respond to a food stimulus. A combination of negative stimulus (herding) and a positive stimulus (low-tone, verbal reassurance) was found effective in keeping the sheep on the exercise program.

The initial jump height (45 cm) was set by the ability of the sheep to easily jump to this level with no weights in the blankets. The initial number of circuits was determined by the sheep's ability to perform each circuit, wearing unweighted blankets, without failing on a jump. The initial blanket weights were set at 12% of live weight.

The experiment

Eight Suffolk-cross ram lambs, ranging in weight from 20 to 25 kg, were paired by weight and randomly assigned to either control or exercised treatments within a pair. The rams were housed in individual pens and were fed a barley/soybean creep feed *ad libitum*

(Table IV.1). Live weight and feed consumption were monitored daily.

Dry matter digestibility was measured using the rare earth element dysprosium (Kennelly *et al.*, 1980; Appendix II). Collection of fecal samples began ten days after the sheep had been given access to the feed containing dysprosium. Grab sample collections were attempted three times per day (early morning, mid-afternoon, and evening) during two consecutive days per week (Saturday and Sunday). Feces collected during the six attempts per week were pooled for each animal. Fecal collections occurred during a two week training period and over the eight week exercise period.

The exercise

An oval track with four platform jumps (including 'down' ramps) was designed (Plate IV.1). Platform jumps with 'down' ramps were used rather than hurdle jumps because the sheep tended to slip when landing on the concrete floor. In addition, the impact of landing on the concrete might have caused injuries, especially when the sheep were carrying lead-weighted blankets. The platform jumps and 'down' ramps were covered with roofing tar and tile which provided a non-slip surface.

The requirements of a progressive-resistance-exercise were fulfilled in three ways (Figure IV.1). The height of the platform was increased by 15 cm after four weeks on experiment. The number of circuits (one circuit equals four jumps) was increased by one circuit every week throughout the experiment. The sheep wore lead-weighted show blankets which were increased by 3% of body weight per week as the experiment progressed.

The only variable which was adjusted on an individual animal basis was blanket weight. Unlike the sheep in the static loading experiments (Chapters II and III), the sheep in this experiment wore the blankets only for the duration of the exercise, not continuously.

Sheep were exercised in groups of two; larger groups tended to cause bunching at the approaches to the jumps and sheep did not work well on their own. After an initial 2 week training period, sheep were exercised five days per week for eight weeks. Sheep were not exercised for 24 hours prior to slaughter.

Table IV.1 The formulation of the pelleted diet which was fed to control and progressive-resistance-exercised sheep.

	Composition (g.kg^{-1}) ^t	Analysis (dry matter basis, g.kg^{-1})	
Alfalfa pellets	400	Crude protein	174
Barley	500	Calcium	10.3
Soybean meal	88	Phosphorous	4.2
		Digestible energy, MJ.kg^{-1}	13.56

^tTo 100 kg of this diet were added 575 g salt, 575 g ground limestone and 20 g Vitamin A-D-E (10,000,000 i.u./kg Vit A, 1,650,000 i.u./kg Vit D, 10,000 i.u./kg Vit E).

Figure IV.1 Increases in platform height, number of circuits, and blanket weight with time on trial

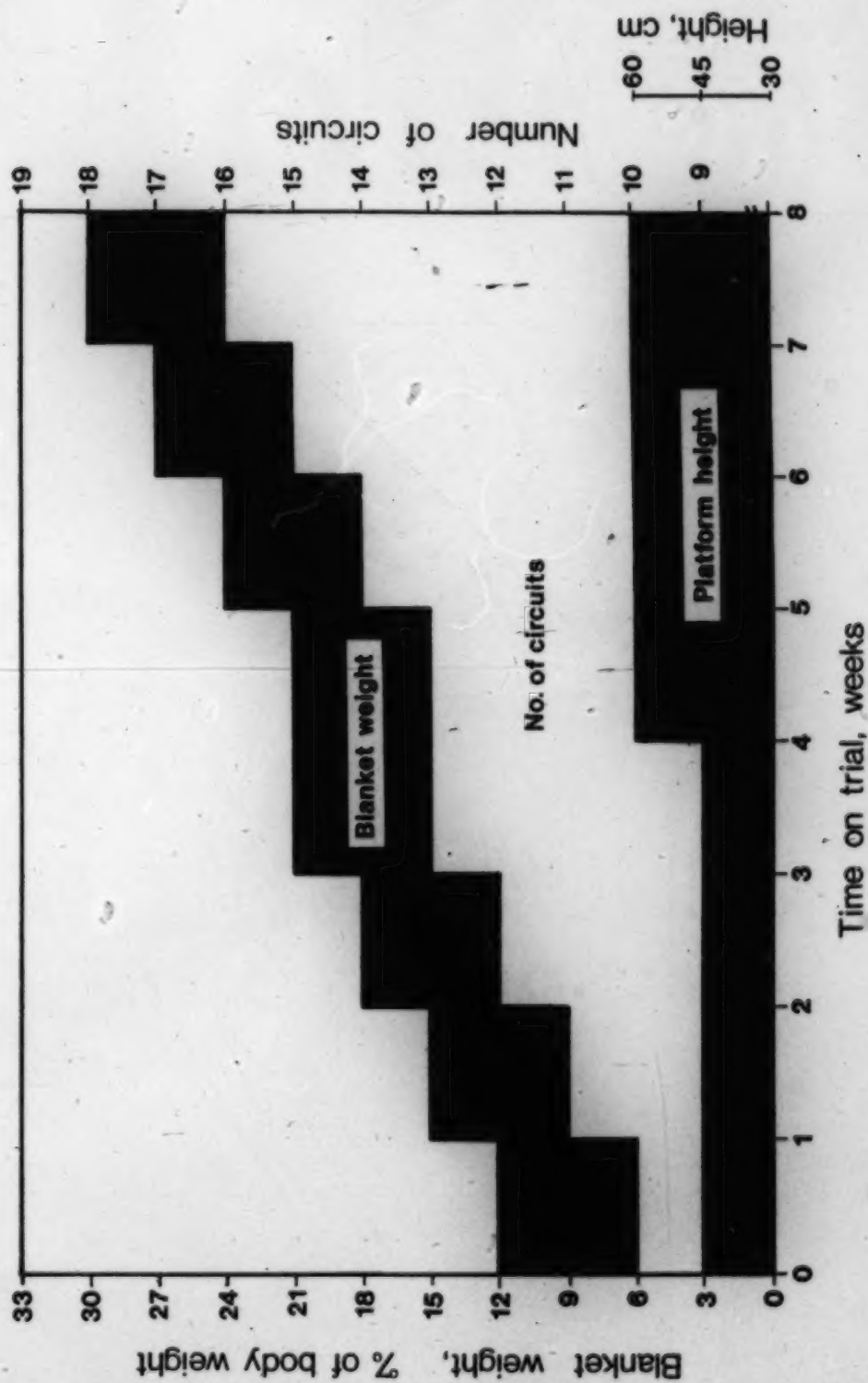




Plate IV.1 The oval jumping circuit

At slaughter

The sheep were 'shrunk' (no feed but free access to water) for 24 hours before slaughter. A final live weight was recorded and the animals were slaughtered and dressed as described in Chapter II. In addition, the full alimentary tract was weighed. The carcass was weighed, split down the backbone, weighed as left and right sides, and the left side was suspended by the Achilles tendon for 48 hours at 6° C. The left side was completely dissected into individual muscles and bones (Butterfield and May, 1966). Fat depot weights were also recorded.

Three 'target' muscles (*vastus lateralis*, *rectus femoris* and *gastrocnemius*) were removed from the left side of the carcass. These muscles were expected to have been preferentially involved in the jumping exercise, due to their dominant role in extension and flexion of the stifle joint and extension of the hock joint (May, 1964).

The *vastus lateralis* was double wrapped in freezer paper and stored at -20° C for subsequent quality analysis (Appendix III). The *rectus femoris* and *gastrocnemius* were measured (length, maximum width) and were bisected at their maximum width. Cross sections of the 'faces' of the bisected muscles were drawn on gridded plastic sheets. Maximum cross-sectional areas were subsequently calculated from the grid.

The same three target muscles were removed at slaughter from the right side of the carcass. Subsamples from these muscles were wrapped and stored at -20° C for proximate analysis (*vastus lateralis*; Appendix IV.A) and myofibrillar protein determination (*rectus femoris* and *gastrocnemius*; Appendix IV.B).

Statistical analysis

A simple one-way analysis of variance was used in most cases. Average daily gains were calculated by regression analysis of live weight with days on trial. SPSSX (1981) was used for statistical analyses. The dysprosium data were analysed using Harvey's program for least squares analysis (Harvey, 1960). The model used was $Y = \mu + A + A/B + C + AC + e$ where A is treatment, B is animals and C is the period (weekly samples pooled per sheep for

10 weeks).

C. RESULTS

The intensity of the progressive-resistance-exercise did not seem equal among all sheep. Two of the exercised sheep appeared to have little difficulty whereas the other two exercised sheep appeared to have moderate difficulty with the initial levels of platform height, number of repetitions and load carried. Although, near the end of the experiment all the exercised sheep had moderate to severe difficulty completing the exercise protocol without resting, the difference in initial intensity between individual sheep may have decreased the overall magnitude of physiological response to the exercise.

As the experiment progressed and the blanket weights became heavier, I noticed that the exercised sheep seemed to be changing their jumping motion. Thus, a picture of the sheep jumping onto the platform was studied to determine the possible contribution of the forelimb muscles to the jumping motion (Plate IV.2). Schematic drawings of the changes in joint angulation and the resulting bone location during the jumping motion are shown in Figure IV.2(a-c).

There were no significant differences in any of the live animal performance data between the control and exercised sheep (Table IV.2). Initial weights were similar by design. Exercised animals had marginally lower average daily gains, which resulted in slightly lower weight gain (23.5 vs. 25.8 kg). Exercised sheep ate less feed resulting in slightly higher feed conversion efficiencies than control sheep. There were no significant differences in dry matter digestibility between exercised and control sheep (Figure IV.3).

There were also no significant differences between exercised and control sheep in the distribution of offal components (Table IV.3) or in the distribution of carcass tissues (Table IV.4).

Although the exercised sheep had a similar total muscle weight as the control sheep (6.7 vs. 6.8 kg), the anatomical distribution of muscle differed (Table IV.5). Exercised sheep had a higher percentage of their muscle weight in the distal thoracic limb than control sheep

Table IV.2 Live animal performance of control and progressive-resistance-exercised sheep.

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Number of animals	4	4	—	—
Initial weight, kg	22.7	22.7	1.2	—
Final weight, kg	48.5	46.3	1.0	0.16
Weight gain, kg	25.8	23.5	0.9	0.18
Average daily gain, $\text{kg} \cdot \text{day}^{-1}$	0.40	0.37	0.01	0.28
Feed consumption, kg	121.1	112.7	4.3	0.22
Feed conversion efficiency, $\text{kg} \cdot \text{kg}^{-1}$	4.7	4.8	0.2	0.66
Time on trial, days	75.0	75.0	—	—

Table IV.3 Offal components (as a percentage of empty body weight[†]) of control and progressive-resistance-exercised sheep.

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Number of animals	4	4		
Empty body weight [†] , kg	39.8	37.4	1.1	0.16
Fleece	14.6	14.5	1.17	0.92
Head	6.3	6.1	0.14	0.55
Front feet	1.05	1.13	0.03	0.17
Hind feet	0.98	1.03	0.03	0.33
Alimentary tract	22.0	23.7	1.91	0.55
Heart	0.51	0.54	0.02	0.24
Lungs	2.3	2.3	0.13	0.85
Liver	2.3	2.3	0.12	0.84
Kidneys	0.31	0.35	0.03	0.41
Spleen	0.20	0.17	0.01	0.10
Bladder	0.04	0.03	<0.01	0.40
Penis	0.24	0.28	0.04	0.51
Testes	0.91	0.96	0.12	0.78

[†] Live weight minus the full alimentary tract weight.

Table IV.4 Half carcass dissection of control and progressive-resistance-exercised sheep.

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Number of animals	4	4	-	-
Dressing percentage †	51.2	51.4	0.70	0.89
Half carcass weight, kg	12.4	11.9	0.43	0.41
Muscle ‡	55.1	56.0	0.83	0.46
Fat ‡	30.8	29.4	1.23	0.44
Bone ‡	13.4	13.8	0.68	0.74
Tendon ‡	0.6	0.8	0.09	0.24
Muscle:bone ratio	4.1	4.1	0.19	0.95

† Warm carcass weight expressed as a percentage of live weight.

‡ Expressed as a percentage of half carcass weight.

Table IV.5 Anatomical muscle group weights (as a percentage of half carcass muscle weight) of control and progressive-resistance-exercised sheep.

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Number of animals	4	4	—	—
Half carcass muscle weight, kg	6.8	6.7	0.2	0.55
Muscle group				
1. Proximal pelvic limb	29.1	28.6	0.47	0.50
2. Distal pelvic limb	4.8	4.8	0.16	0.77
3. Around spinal column	16.5	16.4	0.37	0.86
4. Abdominal wall	11.2	11.1	0.51	0.96
5. Proximal thoracic limb	12.9	13.3	0.19	0.22
6. Distal thoracic limb	3.1	3.3	0.06	0.03
7. Thorax to thoracic limb	8.7	8.8	0.19	0.83
8. Neck to thoracic limb	4.6	4.4	0.19	0.42
9. Neck and thorax	9.0	9.1	0.34	0.78

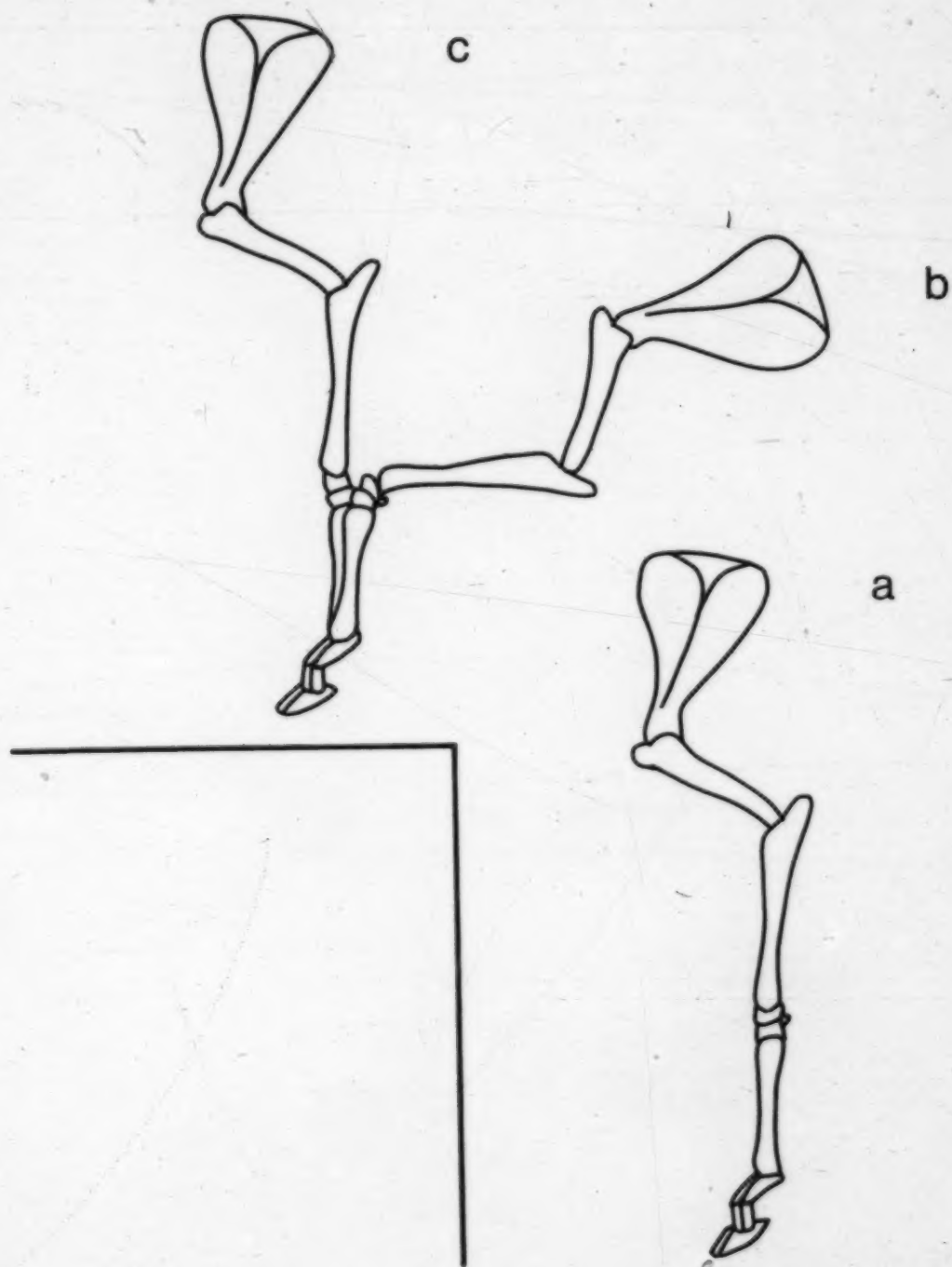


Figure IV.3 Weekly dry matter feed digestibilities of control and exercised sheep

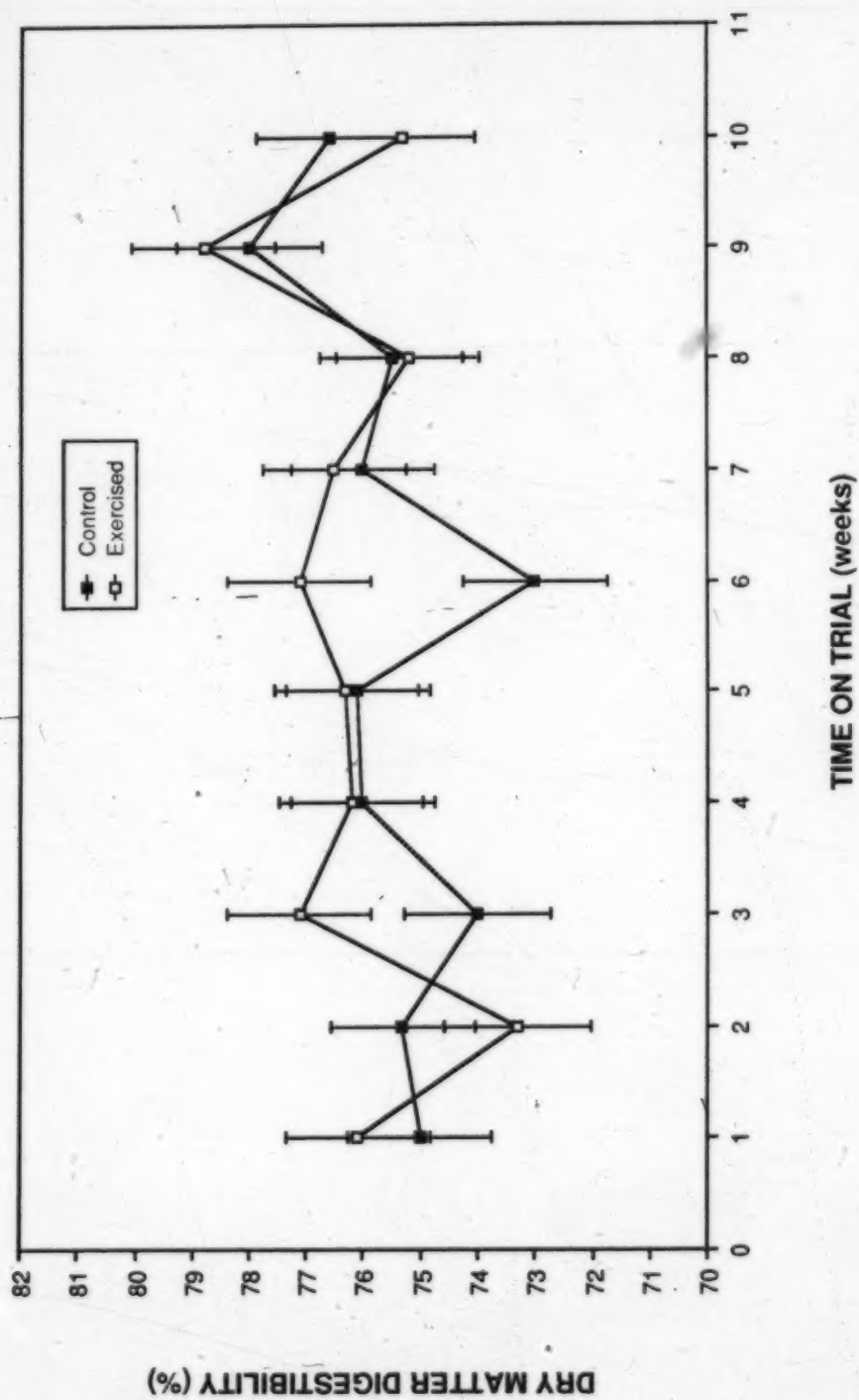




Plate IV.2 Sheep jumping onto the platform

(3.3 vs. 3.1%; $P=0.03$). When individual muscle weights were expressed as a percentage of total muscle weight (Table IV.6), the weights of the *extensor carpi radialis*, *extensor digitorum longus* and *extensor digitorum lateralis* were higher percentages of total muscle weight in the exercised sheep than in the control sheep ($P=0.07$, $P=0.09$, $P=0.09$, respectively; Figure IV.4). Five other muscles in this group also tended to be a higher proportion of total muscle weight in the exercised sheep than in the control sheep.

Weights of two of the muscles in the proximal thoracic limb (Group 5) were also a higher proportion of total muscle weight in the exercised sheep. These were the *biceps brachii* ($P=0.05$) and the *brachialis* ($P=0.04$; Figure IV.4).

There was no significant difference between the exercised and control sheep in the proportion of bone in the appendicular and axial skeletons (Table IV.7). Similarly, there were no significant differences in the weight distribution of individual bones between the exercised and control sheep.

There was no difference between the exercised and control sheep in the partitioning of fat into depots, on either an absolute weight basis or as a percentage of total half carcass fat (Table IV.8).

Thawing and cooking characteristics of the *vastus lateralis* muscle were similar between the exercised and control sheep (Table IV.9). There was no difference in the cooking time required per 100 g of muscle to obtain a final internal temperature of 70° C in muscles from exercised and control sheep.

Samples of the *vastus lateralis* muscle from exercised sheep consistently required less force to shear and to press through the Kramer cell than samples from the control sheep (Figure IV.5). Muscle samples from the exercised sheep had lower Kramer Press-Ground scores than muscle samples from the control sheep (2.02 vs. 2.34 kg.g⁻¹; $P=0.04$). Similarly, muscles from exercised sheep had lower Warner-Bratzler shear scores (4.69 vs. 4.97 kg; $P=0.09$).

Chemical composition of the *vastus lateralis* muscle was not different between the two treatments (Table IV.10). Mean total collagen values measured in the *vastus lateralis* muscle

Table IV.6 Muscle distribution (individual muscle weights as a percentage of half carcass muscle weight) of control and progressive-resistance-exercised sheep.

Muscle Group (- Proximal Pelvic Limb	Treatment I		Treatment II		S.E.M.	Probability
	Control	4	Exercised	4		
Number of animals		4		4		
Half carcass muscle weight, kg		6.8		6.7	±0.2	0.55
1. Tensor fascia latae		1.05		0.97	±0.060	0.34
2. Biceps femoris		5.36		5.11	±0.144	0.27
3. Gluteus medius		3.44		3.26	±0.084	0.18
4. Vastus lateralis		2.33		2.39	±0.070	0.58
5. Gluteus accessorius		0.53		0.59	±0.046	0.41
6. Gluteus profundus		0.34		0.32	±0.021	0.40
7. Rectus femoris		2.27		2.30	±0.086	0.81
8. Semitendinosus		1.86		1.79	±0.055	0.43
9. Gracilis		1.02		1.00	±0.074	0.85
10. Semimembranosus		4.83		4.90	±0.127	0.75
11. Adductor femoris		1.94		1.90	±0.096	0.76
12. Pectineus		0.57		0.54	±0.058	0.68
13. Sartorius		0.24		0.20	±0.018	0.26
14. Gemellus		0.11		0.10	±0.006	0.65
15. Quadratus femoris		0.17		0.21	±0.060	0.70
16. Obturator internus et externus		0.51		0.45	±0.046	0.35
17. Vastus medialis		0.83		0.88	±0.037	0.39
18. Vastus intermedius		0.84		0.88	±0.018	0.17
19. Articularis genu		0.18		0.13	±0.035	0.34
20. Iliacus		0.67		0.73	±0.035	0.25

Continued

Table IV.6 (cont'd.)

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Muscle Group 2 - Distal Pelvic Limb				
1. Gastrocnemius et soleus	1.58	1.61	±0.057	0.75
2. Flexor digitorum superficialis	0.64	0.60	±0.029	0.39
3. Extensor group	0.65	0.69	±0.032	0.48
4. Peroneus longus	0.20	0.22	±0.017	0.44
5. Extensor digiti quarti proprius	0.20	0.19	±0.041	0.81
6. Tibialis anterior	0.23	0.24	±0.033	0.83
7. Tibialis posterior	0.16	0.16	±0.005	0.60
8. Popliteus	0.29	0.28	±0.022	0.71
9. Flexor digitorum longus	0.19	0.19	±0.036	0.97
10. Flexor hallucis longus	0.63	0.66	±0.033	0.45
Muscle Group 3 - Spinal				
1. Psoas minor	0.65	0.60	±0.048	0.49
2. Psoas major	1.56	1.65	±0.032	0.09
3. Quadratus lumborum	0.31	0.30	±0.009	0.23
4. Iliocostalis	0.67	0.73	±0.035	0.25
5. Longissimus	10.23	10.27	±0.450	0.95
6. Spinalis dorsi	1.55	1.48	±0.140	0.74
7. Multifidus dorsi	1.55	1.48	±0.134	0.82
8. Longissimus cervicis	0.25	0.26	±0.034	0.90

Continued

Table IV.6 (cont'd.)

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Muscle Group 4 - Abdominal				
1. <i>Cutaneous trunci</i>	2.04	2.29	±0.228	0.55
2. <i>Serratus dorsalis caudalis</i>	0.21	0.27	±0.052	0.43
3. <i>Obliquus externus abdominis</i>	2.18	2.17	±0.083	0.90
4. <i>Retractor costae</i>	0.17	0.15	±0.013	0.34
5. <i>Obliquus internus abdominis</i>	1.23	1.18	±0.075	0.63
6. <i>Transversus abdominis</i>	1.57	1.56	±0.065	0.85
7. <i>Rectus abdominis</i>	2.57	2.73	±0.178	0.55
8. <i>Diaphragm</i>	1.13	0.78	±0.066	<0.01
Muscle Group 5 - Proximal Thoracic Limb				
1. <i>Deltoides</i>	0.57	0.61	±0.040	0.46
2. <i>Infraspinatus</i>	2.16	2.18	±0.071	0.88
3. <i>Triceps brachii (caput laterale)</i>	0.80	0.79	±0.031	0.80
4. <i>Teres minor</i>	0.26	0.29	±0.024	0.39
5. <i>Triceps brachii (caput longum)</i>	2.58	2.63	±0.099	0.72
6. <i>Tensor fasciae antibrachii</i>	0.37	0.31	±0.012	0.01
7. <i>Supraspinatus</i>	1.97	2.06	±0.050	0.25
8. <i>Biceps brachii</i>	0.63	0.67	±0.011	0.05
9. <i>Teres major</i>	0.54	0.58	±0.025	0.30
10. <i>Coracobrachialis</i>	0.21	0.24	±0.012	0.23
11. <i>Subscapularis</i>	1.08	1.10	±0.032	0.73
12. <i>Brachialis</i>	0.39	0.42	±0.008	0.04
13. <i>Brachiocephalicus</i>	1.14	1.20	±0.056	0.50
14. <i>Triceps brachii (caput mediale)</i>	0.22	0.23	±0.018	0.79

continued

Table IV.6 (cont'd.)

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Muscle Group 6 - Distal Thoracic Limb				
1. Extensor carpi radialis	0.74	0.83	±0.029	0.07
2. Extensor digitorum longus	0.16	0.18	±0.007	0.09
3. Extensor digitorum communis	0.14	0.15	±0.009	0.55
4. Extensor digitorum lateralis	0.15	0.16	±0.005	0.09
5. Extensor carpi ulnaris lateralis	0.38	0.42	±0.020	0.19
6. Abductor pollicis longus	0.08	0.07	±0.011	0.97
7. Flexor carpi radialis	0.14	0.17	±0.009	0.11
8. Flexor carpi ulnaris	0.23	0.27	±0.023	0.28
9. Flexor digitorum sublimis	0.42	0.42	±0.033	0.92
10. Flexor digitorum profundus	0.51	0.54	±0.018	0.24
11. Anconaeus	0.21	0.20	±0.014	0.57
Muscle Group 7a- Thorax to Thoracic Limb				
1. Latissimus dorsi	1.91	1.88	±0.053	0.70
2. Trapezius thoracic	0.54	0.55	±0.036	0.83
3. Serratus ventralis thoracis	1.64	1.74	±0.114	0.59
4. Pectoralis profundus	3.56	3.33	±0.118	0.21
5. Pectoralis superficialis	1.10	1.32	±0.107	0.20

Continued

Table IV.6 (Cont'd.)

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Muscle Group 8 - Neck to Thoracic Limb				
1. Trapezius cervicalis	0.35	0.37	±0.020	0.55
2. Omotransversarius	0.68	0.63	±0.065	0.58
3. Rhomboides	0.70	0.80	±0.034	0.08
4. Serratus ventralis cervicis	2.92	2.62	±0.141	0.18
Muscle Group 9 - Neck and Thorax				
1. Intercostales	2.53	2.63	±0.086	0.48
2. Serratus dorsalis cranialis	0.06	0.07	±0.018	0.63
3. Scalenus dorsalis	0.20	0.27	±0.043	0.30
4. Cervicohyoideus	0.07	0.06	±0.012	0.79
5. Splenius	0.25	0.25	±0.031	0.89
6. Scalenus ventralis	0.50	0.47	±0.041	0.61
7. Longus capitis	0.22	0.23	±0.023	0.81
8. Intertransversarius longus	0.03	0.04	±0.030	0.79
9. Longissimus capitis and atlantis	0.39	0.46	±0.030	0.93
10. Intertransversarii cervicis	0.52	0.46	±0.041	0.32
11. Semispinalis capitis	1.22	1.14	±0.050	0.27
12. Obliquus capitis caudalis	0.50	0.57	±0.084	0.58
13. Rectus thoracis	0.11	0.14	±0.022	0.40
14. Transversus thoracis	0.22	0.23	±0.026	0.87
15. Longus colli	0.71	0.59	±0.046	0.10
16. Multifidi cervicis	0.52	0.51	±0.067	0.94
17. Sternoccephalicus	0.54	0.63	±0.068	0.38

Table IV.7 Bone distribution (individual bone weights expressed as a percentage of half carcass bone weight) of control and progressive-resistance-exercised sheep.

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Number of animals	4	4	—	—
Half carcass bone weight, kg	1.6	1.6	0.08	0.83
Axial skeleton, %				
Cervical vertebrae	47.0	45.9	0.51	0.66
Thoracic vertebrae	7.0	6.2	0.33	0.16
Lumbar vertebrae	9.9	9.1	1.62	0.72
Costae	9.4	9.6	0.65	0.84
Sternum and costal cartilage	13.7	13.5	0.69	0.89
	7.0	7.5	0.67	0.60
Appendicular skeleton, %				
Scapula	52.9	54.0	0.51	0.66
Humerus	4.9	5.1	0.21	0.53
Radio-ulna	7.0	7.2	0.29	0.75
Carpus	5.8	6.1	0.17	0.39
Os coxae	1.0	0.9	0.06	0.86
Femur	10.2	10.8	0.46	0.42
Tibia	9.8	9.9	0.44	0.81
Patella	7.9	8.2	0.29	0.62
Tarsus	0.7	0.6	0.06	0.60
	5.5	5.2	0.23	0.32

Table IV.8 Fat partitioning (absolute fat depot weights and fat depot weights expressed as a percentage of half carcass fat weight) of control and progressive-resistance-exercised sheep.

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Number of animals	4	4		
Half carcass fat weight, kg	3.8	3.5	0.25	0.37
Absolute weight basis, kg				
Subcutaneous fat	1.65	1.60	0.07	0.68
Intermuscular fat	1.30	1.33	0.09	0.83
Perirenal plus channel fat	0.50	0.59	0.06	0.95
Percentage of total half carcass fat, %				
Subcutaneous fat	44.6	43.9	1.93	0.81
Intermuscular fat	35.1	36.4	2.06	0.67
Perirenal plus channel fat	16.5	15.6	1.57	0.71

Table IV. 9 Thawing and cooking characteristics of the *vastus lateralis* muscle from control and progressive-resistance-exercised sheep.

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Number of animals	4	4	-	-
Thawing characteristics				
Fresh weight, g	162	159	7.1	0.79
Thawed weight, g	159	157	6.8	0.80
Percent thaw drip, %	2.1	1.9	0.42	0.72
Cooking characteristics				
Trimmed raw weight, g	144	142	6.2	0.84
Cooked weight, g	112	111	4.2	0.88
Cooking drip, %	3.5	3.6	0.49	0.99
Volatile loss, %	18.5	18.3	0.92	0.89
Cooking time per 100 g, min	23	23	1.53	0.74

Table IV.10 Chemical analysis of the *vastus lateralis* muscle from control and progressive-resistance-exercised sheep.

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Number of animals	4	4	—	—
Water, %	76.2	76.3	0.32	0.84
Protein, % ^f	71.9	73.1	1.77	0.63
Fat, % ^f	15.5	14.3	1.40	0.58
Ash, % ^f	12.6	12.5	0.58	0.92
Total collagen, mg.g ⁻¹ ^f	9.92	7.49	2.71	0.55
Ultimate pH	5.74	5.75	0.04	0.94

^f Expressed on a dry weight basis.

Figure IV.4 The relative locations of the muscles of the forelimb which were a significantly larger porportion of total muscle mass in the exercised sheep

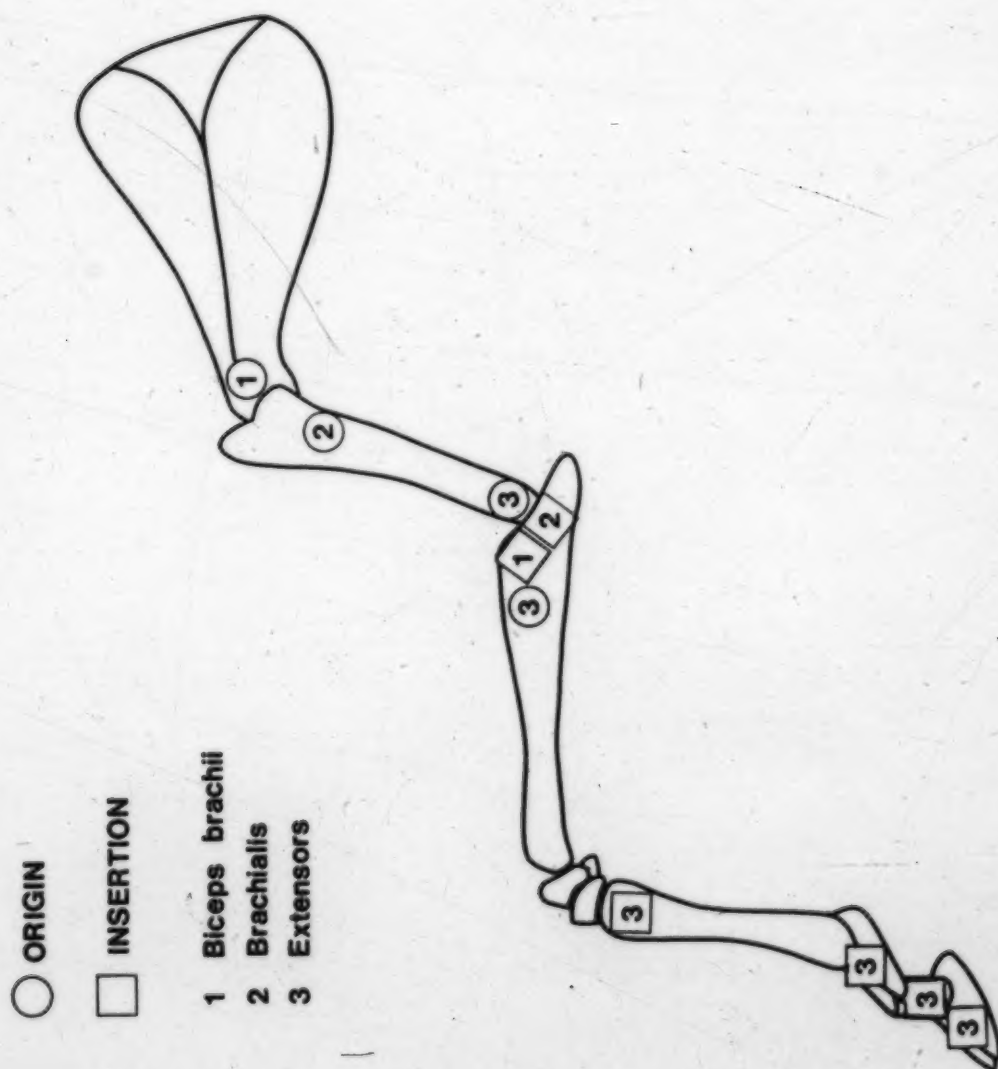
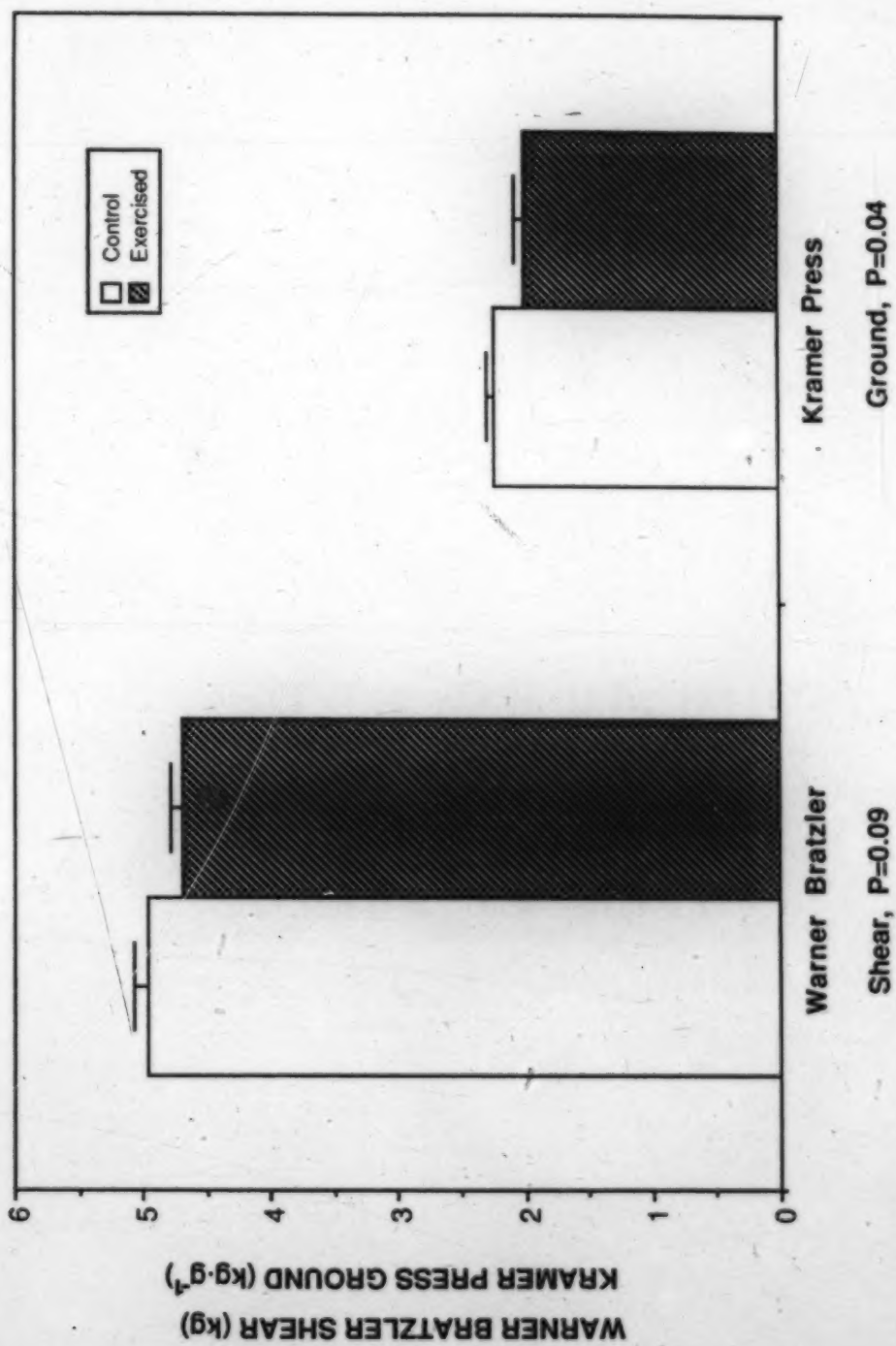


Figure IV.5 Mechanical tenderness tests for the *vastus lateralis* muscle from control and exercised sheep



were much lower in the exercised sheep than in the control sheep (7.49 vs. 9.92 mg.g⁻¹). However, these differences were not significant. Similar ultimate pH values were obtained for both the control and exercised muscles.

Post-mortem muscle shape was not significantly different between the exercised and control sheep (Table IV.11). The *rectus femoris* tended to be slightly longer and narrower in the exercised sheep than in the control sheep, with no resultant change in maximum cross-sectional area. The *gastrocnemius* tended to be slightly longer and wider in the exercised sheep, resulting in a small increase in maximum cross-sectional area.

The myofibrillar protein content (Figure IV.6) was not significantly different between the exercised and control *rectus femoris* and *gastrocnemius* muscles. *Rectus femoris* muscles from control and exercised sheep had 160 and 149 mg of myofibrillar protein per g of frozen muscle, respectively ($P=0.19$). Control and exercised sheep had similar amounts of myofibrillar protein in the *gastrocnemius* muscle (164 vs. 164 mg.g⁻¹; $P=0.97$). *Gastrocnemius* muscles from control and exercised sheep contained on average, more myofibrillar protein per g of muscle than the *rectus femoris* muscles.

D. DISCUSSION

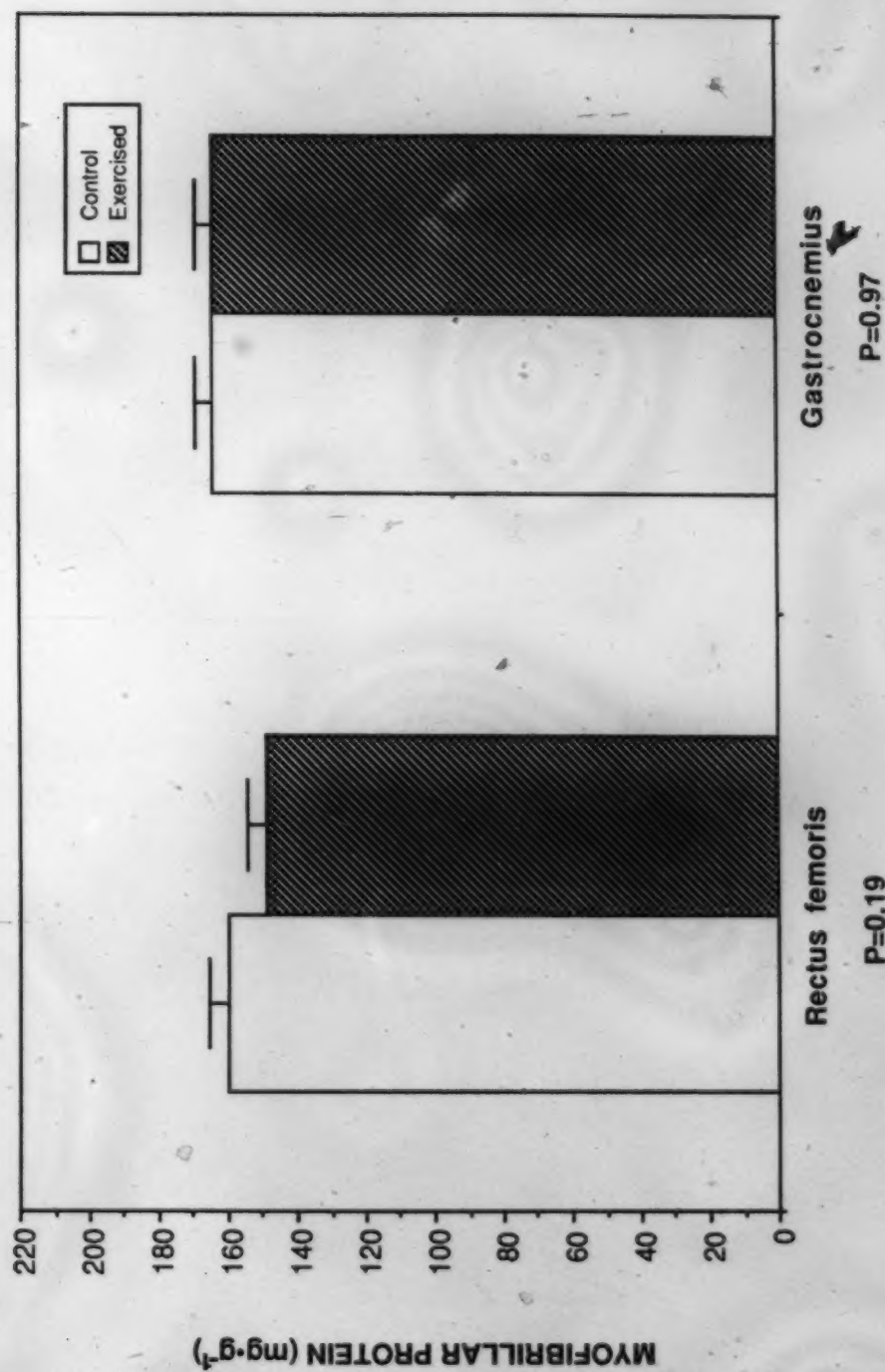
The energy cost of exercise in this experiment was relatively low. Exercised sheep tended to eat less feed (7%) and had slightly lower rates of gain than control sheep, but their performance was generally quite similar. This confirms the data of Morrison *et al.* (1968) and Murray *et al.* (1974a) that the energy cost of exercise is relatively low. Not unexpectedly, the energy cost (in terms of feed conversion efficiencies) of the present progressive-resistance-exercise was smaller than the energy cost of the continuous static loading experiment (Chapter III).

There was no difference between exercised and control sheep in dry matter digestibilities (76.2% vs. 75.6%), even though for six out of the eight week exercise program, the exercised sheep had slightly higher mean dry matter digestibilities than the control sheep. The lack of significant differences was due to large individual animal variation which resulted

Table IV.11 Post-mortem muscle shape characteristics of the rectus femoris and gastrocnemius muscles from control and progressive-resistance-exercised sheep.

	Treatment I Control	Treatment II Exercised	S.E.M.	Probability
Number of animals	4	4	-	-
<i>Rectus femoris</i>				
Muscle weight, g	155	153	6.1	0.87
Length, cm	13.3	13.9	0.31	0.24
Maximum width, cm	6.1	5.7	0.16	0.13
Cross-sectional area, cm ²	20.7	20.1	0.72	0.59
<i>Gastrocnemius</i>				
Muscle weight, g	109	107	5.7	0.86
Length, cm	13.0	13.6	0.34	0.30
Maximum width, cm	5.8	6.0	0.21	0.57
Cross-sectional area, cm ²	17.3	20.3	2.52	0.44

Figure IV.6 Myofibrillar protein content of the *rectus femoris* and *gastrocnemius* muscles
(frozen weight basis) from control and exercised sheep



in a large standard error of the mean.

The dynamic exercise performed in this experiment did not affect bone weight distribution but did result in small changes in muscle distribution. In contrast, bone distribution in the static loading experiments was altered. This provides support for the theory that in static load-carrying exercise much of the increased load is supported by the skeleton whereas, in dynamic exercise, the muscles are used more extensively.

Surprisingly, the muscle distribution data showed that there was no increase in the hindquarter muscles which had been predicted to be heavily involved in the jumping exercise and which had been sampled at the time of slaughter (*vastus lateralis*, *rectus femoris* and *gastrocnemius*). I suspect that this was due to the change in the jumping motion of the sheep which occurred as the blanket weights increased. Initially, the sheep were able to jump onto the platform jumps. However, as the experiment progressed, the sheep began to use the elbow flexor muscles to assist them onto the platform.

The function of the elbow flexors can vary depending on joint angulation in the shoulder and elbow. From the normal standing position, flexion of the elbow joint results in lifting the lower leg. However, when the metacarpal bone and foot are placed perpendicular to the platform (as in this experiment), the radio-ulna forms a joint angle slightly greater than 90° with the metacarpal bone, and the humerus also forms a joint angle slightly greater than 90° with the radio-ulna. With the metacarpal and radio-ulna fixed ahead of the body in this position, flexion of the elbow joint results in a forward movement of the humerus and shoulder joint, drawing the body forward and up onto the platform. This would require a much stronger contraction than merely lifting the lower limb. Apparently, the use of platform jumps, rather than hurdle jumps, allowed the sheep to use their forelimbs to assist their hindlimbs in performing the jumping exercise as blanket weight increased.

In support of this theory, several muscles in the thoracic limb were affected by the exercise; *biceps brachii*, *brachialis*, *extensor carpii radialis*, *extensor digitorum longus* and *extensor digitorum lateralis*. All of these muscles function in flexion of the elbow joint (May, 1964).

The exercise used in this study did result in increased tenderness of the *vastus lateralis* muscle, despite the lack of hypertrophy in that area. This increase in tenderness as a result of both chronic and short-term exercise has been reported previously in the literature.

Exercise immediately before slaughter has been shown to darken the colour, increase the pH, and produce a dry appearance in muscles of hogs (Briskey *et al.*, 1959). Similarly, lambs exercised pre-slaughter had a higher pH and more tender meat in several leg muscles (Chrystall *et al.*, 1982).

The effect of chronic exercise on meat tenderness has been controversial. Mitchell and Hamilton (1933) and Bull and Rusk (1942) found that the tenderness of rib roasts, round steaks and ribs increased as a result of exercise in cattle. Similarly, Spaeth *et al.* (1967) reported an increase in the tenderness of leg muscles from exercised lambs, however, these animals were also treated with diethystilbesterol, which may have affected this result. Mandigo *et al.* (1971) and Hawrysh *et al.* (1974) showed no effect of exercise on tenderness of pork. Hawrysh *et al.* (1974) used the *longissimus* muscle for their determination of tenderness, which was not heavily involved in the treadmill-walking exercise which they had used. Although not stated, it is assumed that Mandigo *et al.* (1971) used the *longissimus* as well. Despite the evidence, there has been a persistent common belief that exercise results in tougher meat.

The mechanism for the increase in meat tenderness resulting from exercise has not been elucidated. However, many factors can contribute to meat tenderness within a breed, at the same chronological age. Some of these factors are: muscle fiber diameter (Smith and Carpenter, 1970), total and soluble collagen content (Dutson *et al.*, 1976; Burson and Hunt, 1986), the amount and rigor state of the myofibrillar protein (Davey and Gilbert, 1969; Bouton *et al.* 1975), the pH and temperature decline post-mortem (Bouton *et al.*, 1982; Yu and Lee, 1986), the activity of proteolytic enzymes (Zeece *et al.*, 1986), the degree of Z-line fractionation post-mortem (Culler *et al.*, 1978) and sarcomere length (McCrae *et al.*, 1971; Hostetler *et al.*, 1972).

In animals exercise-stressed immediately before slaughter, the increase in tenderness is associated with a higher ultimate pH, which influences the physical state of the myofibrillar protein (Briskey *et al.*, 1959; Chrystall *et al.*, 1982). Ultimate pH in both control and exercised *vastus lateralis* muscles were identical in the present experiment. However the rate of pH and temperature decline were not recorded and may have influenced the final rigor state of the myofibrillar protein.

Mitchell and Hamilton (1933) suggested that the increase in tenderness associated with exercise may be due to a slight hypertrophy of the exercised muscle, with no increase in connective tissue. There was no increase in muscle weight or total protein content of the *vastus lateralis* to support muscular hypertrophy. Analysis of the *rectus femoris* and the *gastrocnemius* yielded no differences in myofibrillar protein content. In fact, the *rectus femoris*, which lies beside the *vastus lateralis* in the quadriceps group and performs a similar function, tended to have a lower myofibrillar protein content in muscles from exercised sheep. In addition, there were no significant changes in muscle weight or muscle shape of the *rectus femoris* and the *gastrocnemius* which may have indicated muscular hypertrophy.

The amount of total collagen has a large effect on the tenderness of muscle as measured by mechanical methods (Seidman, 1986). Mitchell and Hamilton (1933) reported a lower percentage of collagen nitrogen in muscles from exercised cattle. Hawrysh *et al.* (1974) did not find a difference in the amount of hydroxyproline between exercised and control pigs in the *biceps femoris* and *psoas major* muscles. Also, Hiner *et al.* (1955) reported that muscles which were used more extensively by an animal had larger amounts of collagenous fibers, whereas those used less extensively had smaller amounts. In this experiment, total collagen content of the *vastus lateralis* was not significantly different between exercised and control sheep. However, the difference in the means may be an indication that a difference did exist; exercised sheep tended to have a lower mean collagen content than control sheep. The low number of sheep used in this experiment, combined with the high degree of individual variation in muscle collagen content contributed to the high standard error of the mean.

E. CONCLUSION

As predicted, the more dynamic progressive-resistance-exercise performed in this experiment did affect muscle distribution more than bone distribution, but had little effect on total muscle weight. The elbow flexors (*biceps brachii*, *brachialis*, *extensor carpi radialis*, *extensor digitorum longus* and *extensor digitorum lateralis*) were a slightly larger proportion of total muscle mass in the exercised sheep due to their involvement in "pulling" the sheep up onto the platform jumps. Unfortunately, the elbow flexors were not the muscles which had been predicted to be most affected by the exercise and were not sampled at slaughter.

Muscle samples taken from the hindlimb were more tender in exercised sheep than in control sheep. The reason for this difference was not apparent but it did not appear to be caused by hypertrophy of the exercised muscle or an increase in the myofibrillar protein content. The amount of total collagen may have contributed to the increase in tenderness but the results were inconclusive. In addition, there are many other factors which can influence meat tenderness which were not investigated.

Chapter V

THE EFFECTS OF ENDURANCE EXERCISE ON GROWTH, DEVELOPMENT AND MEAT QUALITY IN SHEEP

A. INTRODUCTION

Small changes in muscle distribution occurred in response to the progressive-resistance jumping exercise in the previous experiment (Chapter IV). These types of changes, although of physiological interest, are of little potential benefit to meat production. Of greater potential value to meat production was the increase in tenderness of the meat from exercised sheep. Although this result has been reported previously in studies of meat animals (Mitchell and Hamilton, 1933; Bull and Rusk, 1942; Spaeth *et al.*, 1967), the underlying cause for the increase in meat tenderness in exercised animals has not been determined.

One of the major problems encountered in the previous experiment was the difference in the abilities of the sheep to perform the exercise. Differences in the ability to perform an exercise can result from many factors at the systemic (heart rate, stroke volume), cellular (capillary density, fiber type distribution, neuromuscular junctions) and subcellular (enzyme activity, energy stores) levels (Fox and Mathews, 1981). The oxygen consumed to perform a certain exercise load is an indication of the intrinsic ability to perform the exercise. In studies of human exercise, an individual's maximum oxygen consumption ($\text{VO}_2 \text{ max}$) for a given exercise is used to set individual exercise intensities.

The following experiment was designed to examine the effect of an endurance exercise performed at 75% $\text{VO}_2 \text{ max}$ on meat quality and related cellular and subcellular components of sheep. The effects of endurance exercise on live animal performance, body composition and tissue distribution were also investigated.

B. MATERIALS AND METHODS

The experiment

Twenty-two Suffolk ram lambs (27-36 kg) were pair-weighted and each pair assigned to one of the following weight classes: heavy, medium or light. Animal pairs were randomly assigned within each weight classification to one of three slaughter groups (after 6, 12 and 18 weeks of exercise). Within each pair, sheep were randomly assigned to either control or exercised groups. Each pair within a slaughter group was stagger-started on experiment so that only one pair would be ready for slaughter each week. The sheep were fed a pelleted alfalfa/barley/soybean meal ration *ad libitum* (Table V.1). To monitor feed consumption, two sheep of the same treatment were housed in each pen.

The exercise

Sheep were exercised on a single belt treadmill driven by an electric variable speed motor. Oxygen consumption was monitored using a Beckman OM-11 Oxygen Analyzer in a continuous flow system (Appendix V). The sheep used only a walking or trotting gait on the treadmill, depending on treadmill speed. There were two problems that we encountered while using the treadmill. First, the exercised sheep panicked if they were put on the treadmill alone. This problem was solved by putting the corresponding control sheep into a stall in front of, and facing the exercising sheep. The two sheep were separated by a plexiglass divider. The second problem was encountered when the exercised sheep urinated on the treadmill. Since the treadmill belt had no tread, it became very slippery when wet. This problem was solved by building urine collectors which directed the urine into a bottle at the side of the treadmill without interfering with the sheep's stride. Emergency supplies of sawdust were kept on hand to soak up moisture in case of accidental spills. Once these problems were solved, the sheep were allowed two weeks to acclimatize to the treadmill and to the flow-through oxygen masks.

Table V.1 The formulation of the pelleted diet which was fed to control and endurance-exercised sheep.

	Composition (g.kg ⁻¹) [†]	Analysis (dry matter basis, g.kg ⁻¹)	
Barley	504	Crude protein	163
Alfalfa	400	Calcium	8.0
Soybean meal	89	Phosphorus	3.0
		Digestible energy, MJ.kg ⁻¹	17.22

[†]To 100 kg of this diet were added 600 g salt, 30 g Vitamin A-D-E (10,000,000 i.u./kg Vit A, 1,650,000 i.u./kg Vit D, 10,000 i.u./kg Vit E).

One week prior to the start of training, two VO_2 max tests were conducted for each sheep (including controls). The VO_2 max test consisted of a 5 minute warm-up at 2.4 kilometers per hour (kmph), followed by 5 minutes of rest (standing). Animals then resumed exercising at 3.4 kmph for three minutes followed by incremental increases (4.5 kmph, 5.1 kmph, 6.1 kmph, 7.2 kmph) every three minutes, without rest, until exhaustion. Exhaustion was reached when the oxygen decrement either peaked or reached a plateau (Figure V.1). In cases where the oxygen decrement did not peak or reach a plateau, exhaustion was judged visually when the sheep tripped, rode back on the retaining bar or skidded on the treadmill. Training speeds were set to maintain an oxygen decrement equivalent to 75% VO_2 max for the exercised animals. Training speeds were adjusted weekly, based on daily measurements of oxygen consumption (at rest and during exercise), to maintain workloads of 75% of VO_2 max. Every three weeks, exercised animals were re-tested to determine changes in oxygen consumption and VO_2 max. Exercised sheep were worked on the treadmill five days per week (Tues, Weds, Fri, Sat, Sun) and length of time on the treadmill was increased during the experiment to a maximum of 90 minutes per day for sheep in the third slaughter group (Figure V.2).

At slaughter

Four days before slaughter, exercised sheep were given a day off exercise; a day later, the exercised sheep performed a final VO_2 max test. The next day the animals were not exercised, but were fed *ad libitum*. Twenty-four hours prior to slaughter the exercised and control pair were removed from their respective pens and placed in a pen together, without feed but with free access to water.

A final live weight was obtained just prior to slaughter. Sheep were stunned with a captive bolt pistol and killed by jugular/carotid exsanguination. The bodies were dressed in the normal commercial manner and the offal components were weighed (described in Chapter II) with the following exceptions: the empty heart was weighed, trimmed of fat, and re-weighed; the full alimentary tract and testes were weighed. The carcass was split medially and right and left sides were weighed.

Figure V.1 Typical oxygen decrement during VO_2 maximum testing

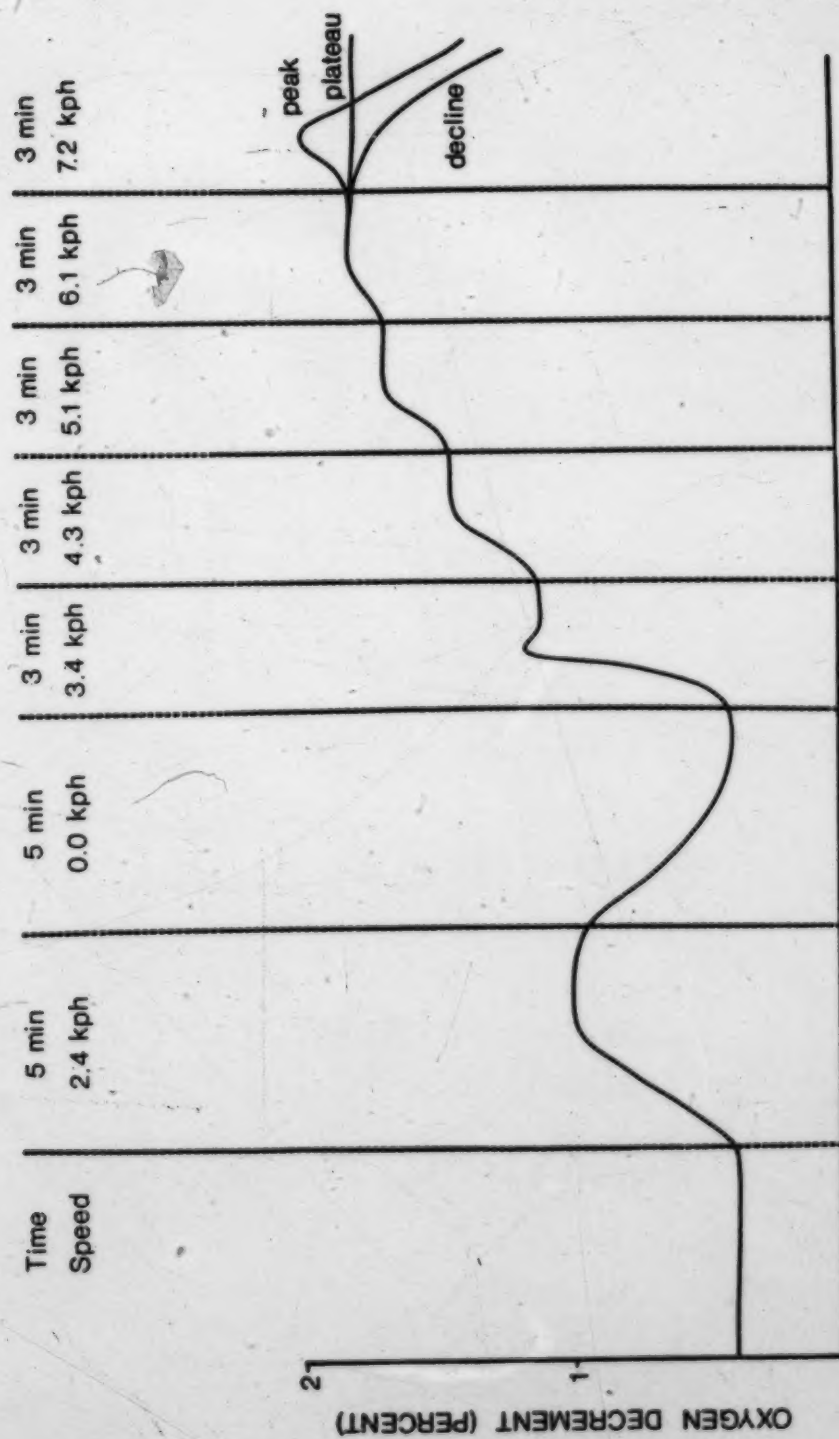
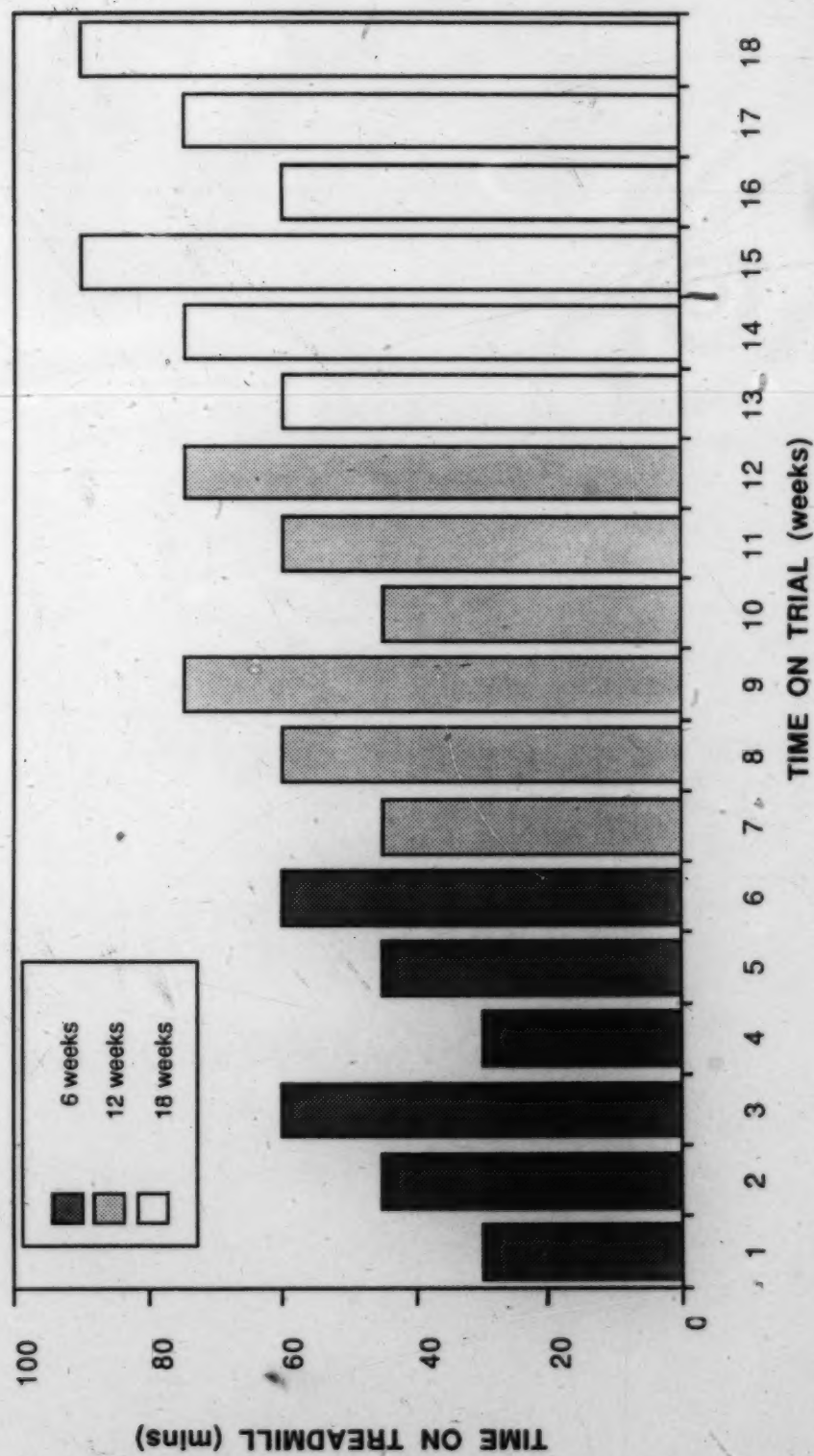
VO₂ MAXIMUM TEST

Figure V.2 The increase in daily time on treadmill with time on trial



Five muscles (*semimembranosus*, SM; *vastus lateralis*, VL; *semitendinosus*, ST; *psaos major*, PM; and *gastrocnemius*, G) were removed from the right side of the carcass, trimmed of fat and connective tissue and weighed. These muscles were chosen for their range in fiber type (ST vs. PM), range in involvement in treadmill walking (G vs. PM), previous use for mechanical tenderness tests (VL, Chapter IV) and size, fiber direction and function (SM). Samples were removed from all five muscles for the following histological and chemical analyses (Figure V.3):

1. **Histology (Appendix I)**

- a. Myosin ATPase (Guth and Samaha, 1970; Appendix I.B)
- b. NADH dehydrogenase (Bancroft and Stevens, 1977; Appendix I.C)

2. **Chemical (Appendix IV)**

- a. Proximate analysis (AOAC, 1980; Appendix IV.A)
- b. Protein fractionation (Perry and Corsi, 1958; Appendix IV.B)
- c. Total and soluble collagen (Stegemann and Stalder, 1967; Culler et al., 1978; Appendix IV.C)
- d. Myofibrillar fragmentation index (Davey and Gilbert, 1969; Culler et al., 1978; Appendix IV.D)

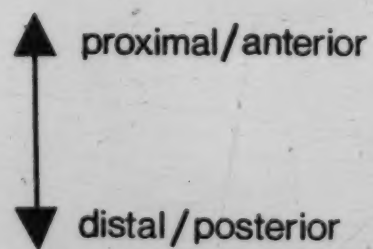
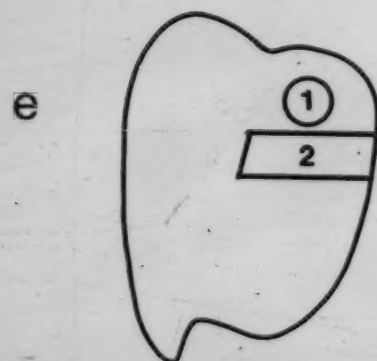
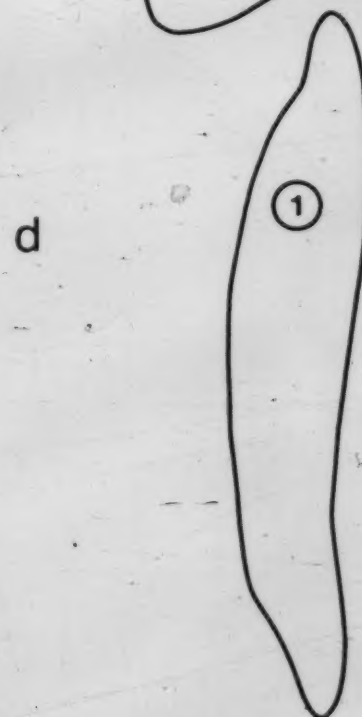
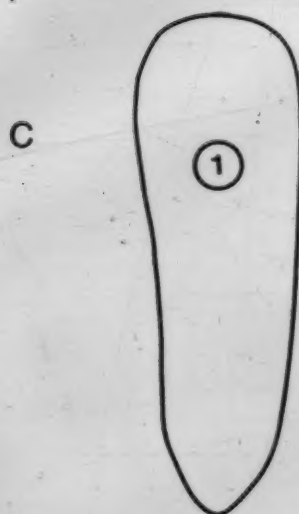
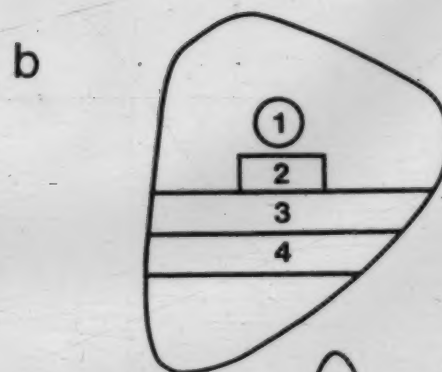
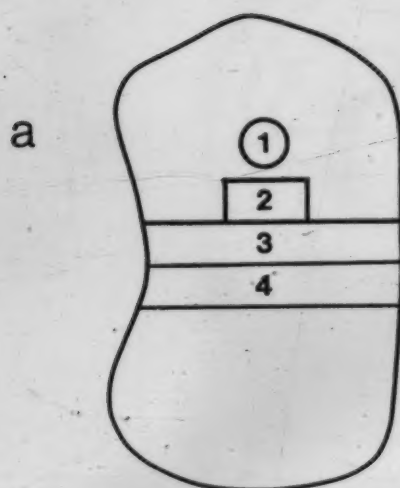
The left side was chilled for 48 hours at 6° C. The pH and temperature of the same five muscles were monitored with a Philips puncture probe at 1, 2, 4, 7, 13 and 25 hours post-mortem (Appendix VI). After 48 hours, the left side was completely dissected as described in Chapter II. The SM, VL, ST, PM and G were weighed and subsampled as follows (Figure V.4):

1. **Meat Quality (Appendix III)**

- a. Linear measurements (Appendix III.A)
- b. Thawing and cooking losses (Appendix III.B)
- c. Warner Bratzler Shear (Appendix III.C)
- d. Kramer Press - Ground (Appendix III.C)
- e. Ultimate pH (Appendix III.D)

- 
1. fiber typing
 2. protein fractionation
 3. total and soluble collagen
 4. myofibrillar fragmentation
 - remaining muscle used for proximate analysis

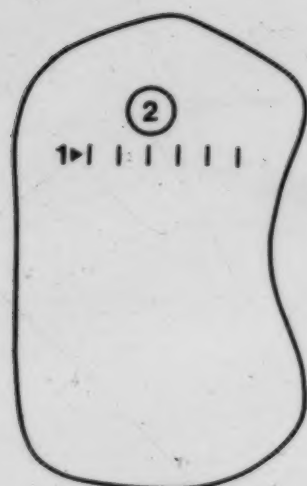
Figure V.3 Post-slaughter sampling sites for various histological and chemical analyses on the right a) *semimembranosus*, b) *vastus lateralis*, c) *semitendinosus*, d) *psoas major* and e) *gastrocnemius* muscles



1. pH and temperature readings
2. sarcomere length and fiber diameters
3. residual glycogen

Figure V.4 Post-mortem sampling sites for various meat quality, chemical and microscopic analyses on the left *a) semimembranosus, b) vastus lateralis, c) semitendinosus, d) psoas major* and *e) gastrocnemius* muscles

a



b



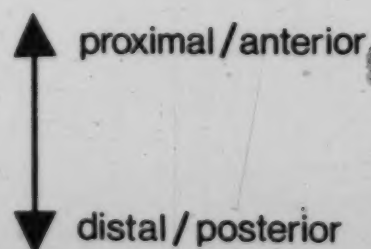
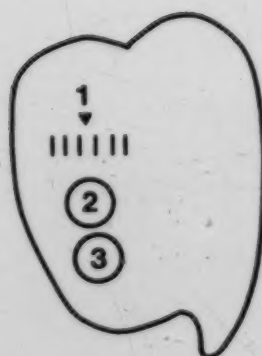
c



d



e



2. Chemical (Appendix IV)
 - a. Glycogen (Lo et al., 1970; Appendix IV.E)
3. Microscopy (Appendix VII)
 - a. Muscle fiber diameters (Swatland, 1980; Appendix VII.B)
 - b. Sarcomere lengths (Swatland, 1980; VII.C)

Statistical analysis

All data were analysed using a two-way analysis of variance, with treatment and slaughter group as the main factors. Slaughter group means were separated at, or below, the 5% level of significance using the Student-Newman-Keuls (SNK) multiple range test (Steel and Torrie, 1980). Treatment by slaughter group interaction means were reported when significant.

Exercised sheep were tested for resting and maximum oxygen consumptions every three weeks, including pre-trial tests; thus, animals in the last slaughter group went through seven oxygen consumption test periods. Resting and maximum oxygen consumption data were expressed in Watts per unit of metabolic body weight ($BW^{.75}$) and their difference was calculated as net oxygen consumption. Analysis of the data was performed using a repeated measures analysis where animals and periods were the main factors. This analysis was used to test two models: (1) over 5 periods, using 8 animals (animals from both the 12 and 18 week slaughter groups); (2) over 7 periods, using 4 animals (animals from only the 18 week slaughter group).

A covariate analysis of the carcass tissue weights was performed to determine if initial weight contributed significantly to the variance. Initial weight was significantly correlated with slaughter group for total and intermuscular fat at the 5% level of probability. However, the adjustment for variations in initial weight had little effect on the slaughter group probabilities, so the data were left unadjusted.

Average daily gains were calculated as the regression of weight on time (0-6 weeks, 6-12 weeks, 12-18 weeks) for each animal within a slaughter group. A two-way analysis of

variance was used to test for treatment and slaughter group differences. Significant slaughter group means were separated at, or below, the 5% level of probability using the SNK multiple range test.

Since the relationship of temperature and pH with time post-mortem is curvilinear, various transformations of the pH and temperature data were attempted to obtain the best fit rectilinear regressions. The best fit for pH and temperature with time was obtained with time expressed as time^{0.1}. The best fit for pH with temperature was obtained with untransformed data. Least squares analysis of variance (Harvey, 1960) of the pH data, with either the transformed time or untransformed temperature as a covariate, was performed with treatment and slaughter group as main factors.

Analysis of least square means with unequal cell sizes (UANOVA) were used to analyse the fiber type data which were expressed as either a frequency or a percentage. Main factors in the model were fiber type, treatment and slaughter group. Fiber type had three levels (glycolytic, oxidative and oxidative-glycolytic) for the NADH dehydrogenase stain and two levels (slow and fast) for the alkaline myosin ATPase stain.

Sarcomere lengths were analysed using a repeated measures analysis (ANOVAR). The main factors in the analysis were treatment, slaughter group and muscle (5 levels). Sarcomere lengths for each muscle were tested using a covariate analysis to determine if carcass weight contributed significantly to the variance.

Descriptive statistics of the fiber diameter frequency distribution (mean, standard deviation, skewness and kurtosis) were analysed using a two-way analysis of variance on individual muscles. Since sarcomere lengths were significantly shorter in exercised sheep than in control sheep, muscle fiber diameters and standard deviations were adjusted using the overall mean sarcomere length for each muscle (Swatland, 1980). Descriptive statistics for the frequency distribution of the adjusted fiber diameters were analysed using a two-way analysis of variance. A four factor least squares analysis of the unadjusted and adjusted descriptive statistics with unequal cell sizes was used to test for significant adjustment differences. The four main factors in this analysis were treatment, slaughter group, muscle

and adjustment.

SPSSX (1980) and two user-defined procedures within SPSSX, developed by the University of Alberta (UANOVA and ANOVAR) were used for statistical analyses.

C. RESULTS

Oxygen consumption

Maximum oxygen consumption and resting oxygen consumption of exercised sheep appeared to decrease with time on trial (Figure V.5). Net oxygen consumption did not change. There was a significant decline in resting oxygen consumption with time and no change in maximum oxygen consumption with time using either statistical model (Figure V.6). There was a significant difference among animals for both maximum oxygen consumption ($P=0.01$) and net oxygen consumption ($P=0.005$) using Model #1 (8 animals over 5 periods). However, there was no animal effect using Model 2 (4 animals over 7 periods).

Live animal performance

Initial weights of the sheep were similar between treatments and among slaughter groups by design (Table V. 2). Although exercised sheep ate significantly more feed than control sheep (285.1 vs. 268.0 kg), they did not differ in final weight, weight gain or efficiency of feed conversion. As expected, final weight, weight gain and feed consumption were significantly different among slaughter groups. The average feed conversion efficiency was not significantly different among slaughter groups.

There was no significant difference between treatments in average daily gain, although control sheep had slightly lower average daily gains for all three exercise periods (0-6 weeks, 6-12 weeks, 12-18 weeks; Figure V.7). The 6 week slaughter group had a significantly larger average daily gain than either the 12 or 18 week slaughter groups (0.418 vs. 0.334, 0.308 kg.day⁻¹; $P=0.03$). As expected, the y-intercepts (live weight) were significantly different

Table V.2 Live animal performance of control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		Probability
	Control	Exercised	
Number of animals	11	11	-
Initial weight, kg	67.1±1.76	67.8±1.76	-
Final weight, kg	93.2±2.14	96.6±2.14	0.28
Weight gain, kg	26.0±1.82	28.8±1.82	0.31
Feed consumption, kg	268.0±4.64	285.1±4.64	0.02
Feed conversion efficiency, kg.kg ⁻¹	10.9±0.77	10.0±0.77	0.43
Slaughter Group			
	6 Weeks	12 Weeks	18 Weeks
Number of animals	6	8	8
Initial weight, kg	65.2±2.38	69.5±2.06	68.3±2.06
Final weight, kg	80.3±2.89 ^a	94.5±2.51 ^b	106.4±2.51 ^c
Weight gain, kg	15.1±2.56 ^a	26.0±2.22 ^b	38.1±2.22 ^c
Feed consumption, kg	146.9±6.29 ^a	263.3±5.44 ^b	387.0±5.44 ^c
Feed conversion efficiency, kg.kg ⁻¹	10.0±1.04	10.7±0.90	10.5±0.90
			0.86

a,b,c Means not followed by the same letter are significantly different at the 5% level of probability. There were no significant treatment by slaughter group interactions for any of these traits.



Figure V.5 Oxygen consumptions at rest and during VO_2 maximum testing and calculated net oxygen consumption (over all endurance-exercised sheep)

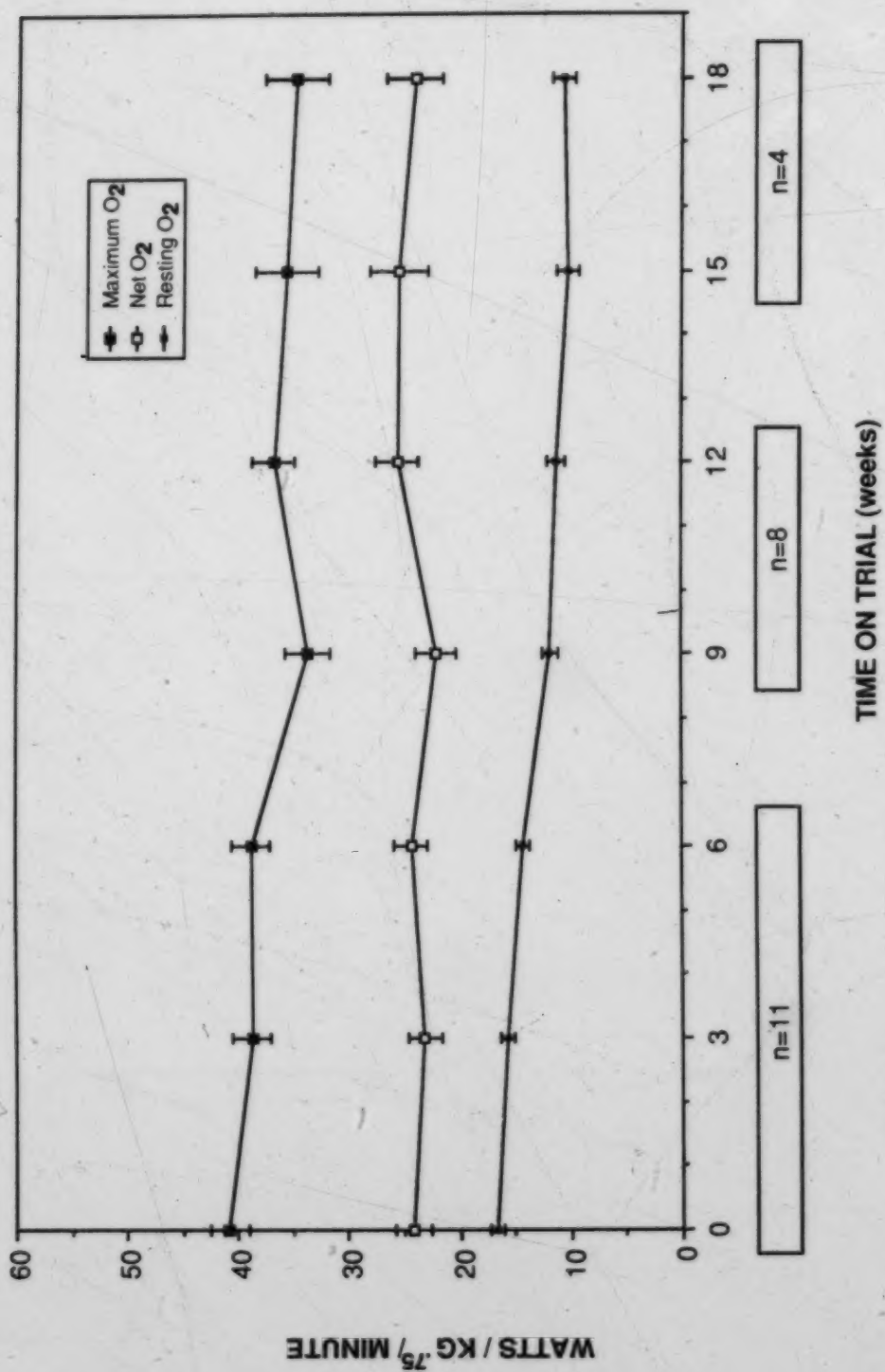


Figure V.6 Decrease in resting oxygen consumption of endurance-exercised sheep over time (2 models)

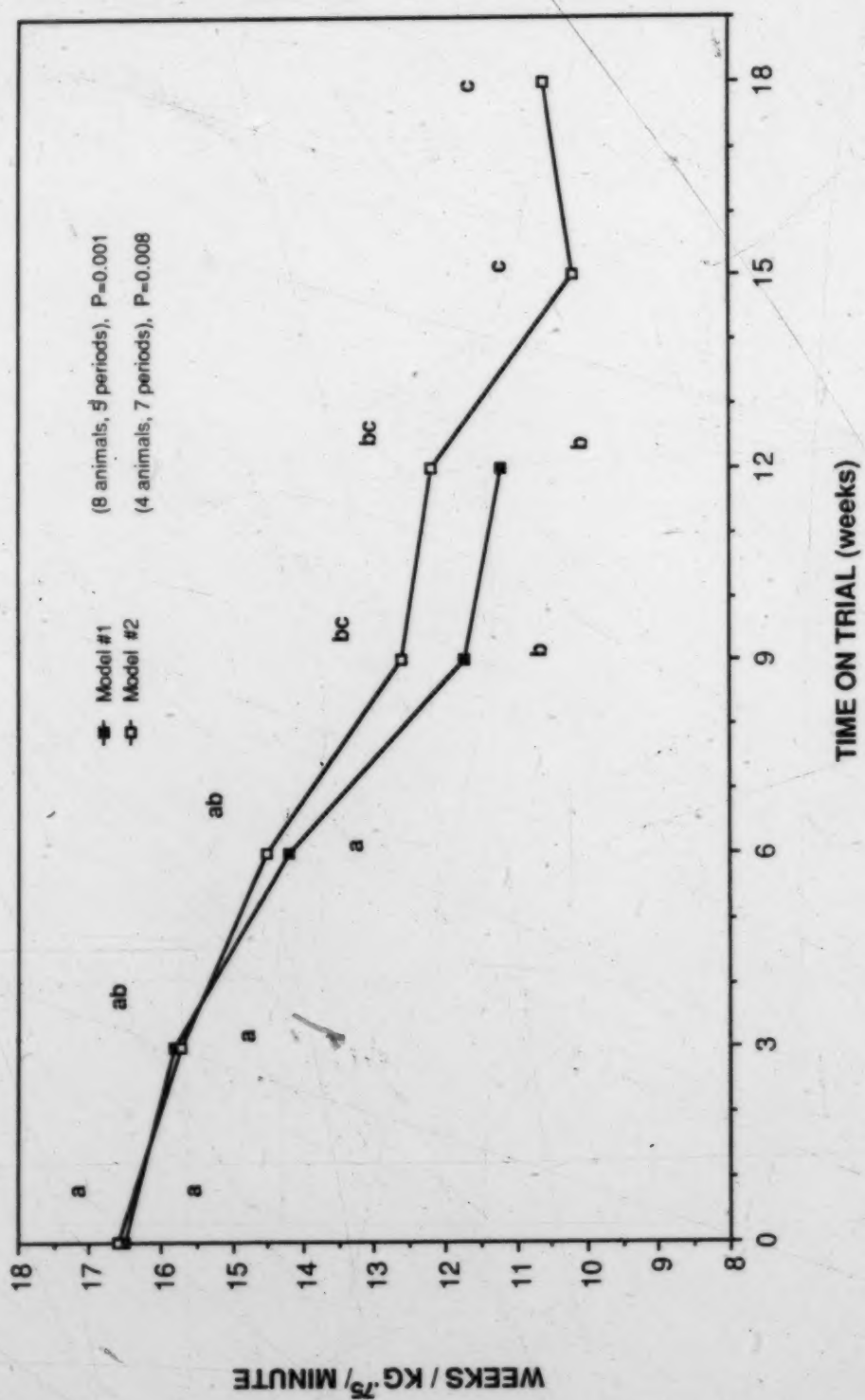
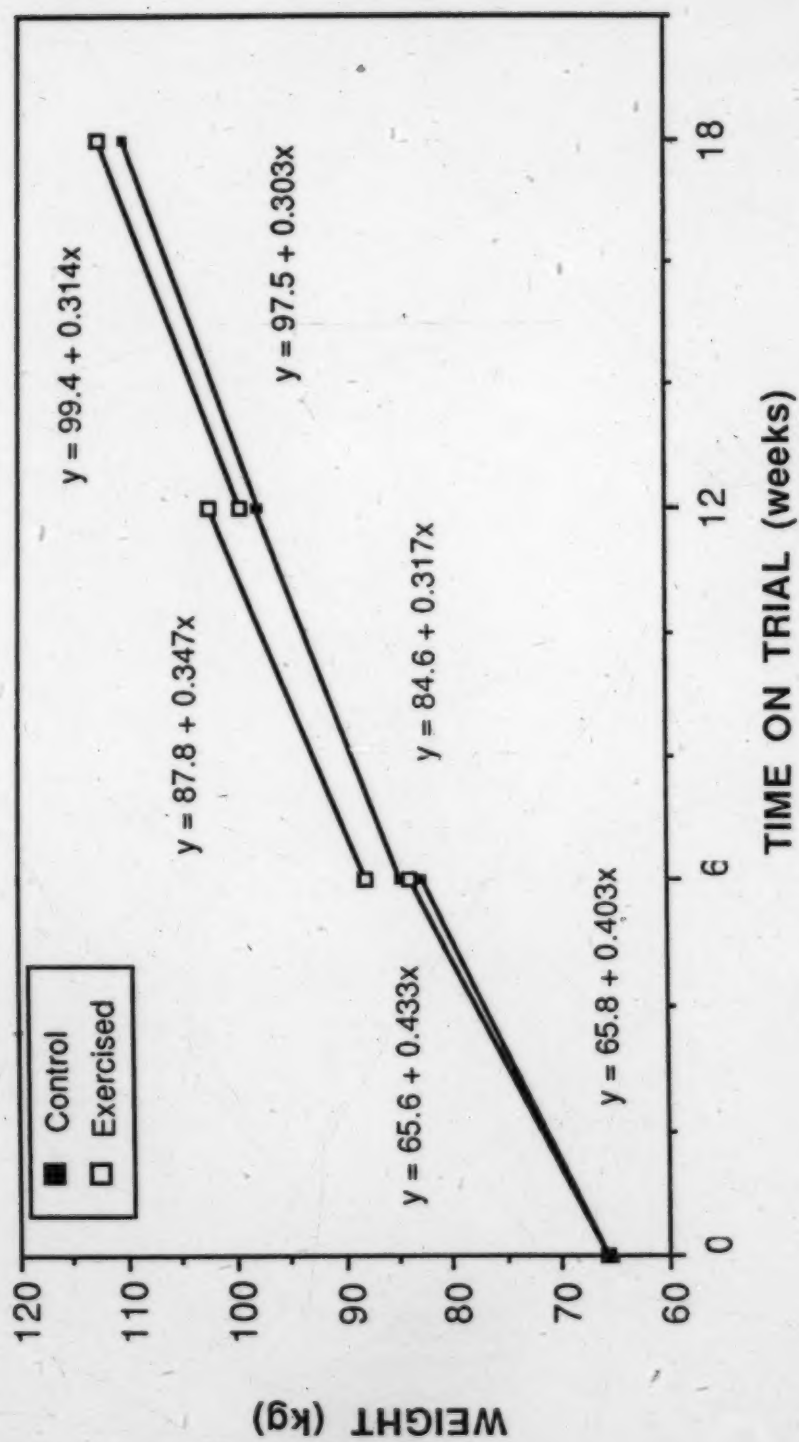


**Figure V.7 Average daily gains of control and endurance-exercised sheep split by treatment
and by slaughter group**



among slaughter groups.

Offal components

There were no significant differences between exercised and control sheep in the proportions of offal components (Table V.3). However, exercised sheep did tend to have a slightly larger proportion of fleece ($P=0.08$) and a slightly larger proportion of liver ($P=0.09$). These differences still tended to exist when expressed as absolute weights. Among slaughter groups (Table V.4), as empty body weight increased from 66.7 kg in the 6 week slaughter group to 86.5 kg in the 18 week slaughter group, the proportion of heart, liver and kidneys decreased significantly ($P<0.01$, $P<0.01$, $P=0.04$, respectively). The proportion of hind feet also tended to decrease over the three slaughter groups ($P=0.06$).

Carcass characteristics

Exercised sheep were not significantly different from control sheep with respect to any of the half carcass dissection data, with the exception of the proportion of tendon (Table V.5). Exercised sheep had a lower proportion of dissectible tendon than controls (0.8 vs. 1.0%; $P=0.05$), primarily due to a much lighter *ligamentum nuchae* weight in the exercised sheep (47.7 vs. 73.0 g; $P=0.04$). As expected, half carcass weight increased over the three slaughter groups (21.7 vs. 25.7 vs. 28.7 kg; $P<0.01$). As half carcass weight increased, the proportion of fat also increased and the proportions of bone and muscle decreased.

Muscle distribution

Total muscle weight did not differ between exercised and control sheep (Table V.6). However, there were several significant treatment differences in the proportion of muscle in the anatomical muscle groups. Exercised sheep had a significantly higher proportion of muscle in the proximal pelvic limb and in the abdominal wall (Groups 1 and 4) than control sheep ($P=0.02$, 0.06 , respectively). Accompanying these increases was a decrease in the proportion of muscle surrounding the spinal column (Group 3) in the exercised sheep. In

Table V.3 Offal components (expressed as a percentage of empty body weight[†]) of control and endurance-exercised sheep split by treatment.

	Treatment		Probability
	Control	Exercised	
Number of animals	11	11	
Empty body weight [†] , kg	76.4±2.22	78.4±2.22	0.61
Fleece	10.97±0.27	11.68±0.27	0.08
Head	4.91±0.15	4.57±0.15	0.13
Front feet	1.10±0.04	1.11±0.04	0.87
Hind feet	0.98±0.029	0.98±0.029	0.96
Alimentary tract	21.70±1.84	23.25±1.84	0.56
Heart	0.51±0.032	0.55±0.032	0.43
Lungs	0.93±0.040	0.92±0.040	0.89
Liver	1.77±0.056	1.91±0.056	0.09
Kidneys	0.27±0.011	0.29±0.011	0.37
Spleen	0.16±0.009	0.17±0.009	0.48
Bladder	0.26±0.089	0.12±0.089	0.30
Penis	0.24±0.031	0.23±0.031	0.87
Testes	1.10±0.059	0.97±0.059	0.16

[†] Live weight minus the full alimentary tract weight.

There were no significant treatment by slaughter group interactions for any of these traits.

Table V.4 Offal components (expressed as a percentage of empty body weight¹) of control and endurance-exercised sheep split by slaughter group.

	Slaughter Group			Probability
	6 Weeks	12 Weeks	18 Weeks	
Number of animals	6	8	8	-
Empty body weight ¹ , kg	66.7±3.01 ^a	76.7±2.60 ^b	86.5±2.60 ^c	<0.01
Fleece	11.12±0.36	11.19±0.31	11.61±0.31	0.53
Head	4.51±0.20	4.76±0.18	4.90±0.18	0.38
Front feet	1.13±0.06	1.11±0.05	1.07±0.05	0.70
Hind feet	1.01±0.04	1.02±0.03	0.91±0.03	0.06
Alimentary tract	21.12±2.49	23.04±2.16	22.93±2.16	0.82
Heart	0.68±0.043 ^a	0.46±0.037 ^b	0.48±0.037 ^b	<0.01
Lungs	0.98±0.055	0.94±0.047	0.86±0.047	0.26
Liver	2.05±0.075 ^a	1.86±0.065 ^a	1.65±0.065 ^b	<0.01
Kidneys	0.31±0.015 ^a	0.28±0.013 ^{a,b}	0.25±0.013 ^b	0.04
Spleen	0.17±0.012	0.17±0.010	0.16±0.010	0.67
Bladder	0.13±0.120	0.15±0.104	0.28±0.104	0.57
Penis	0.29±0.042	0.23±0.036	0.19±0.036	0.26
Testes	1.02±0.080	0.96±0.070	1.12±0.070	0.31

¹ Live weight minus the full alimentary tract weight.

a,b,c Means not followed by the same letter are significantly different at the 5% level of probability.

There were no significant treatment by slaughter group interactions for any of these traits.

Table V.5 Half carcass dissection of control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		Probability
	Control	Exercised	
Number of animals	11 ^f	11 ^a	-
Half carcass weight, kg	25.3±0.64	26.1±0.64	0.36
Dressing percentage [†]	55.6±0.46	55.8±0.46	0.75
Muscle [‡]	49.1±0.79	48.3±0.79	0.51
Fat [‡]	36.6±0.93	37.9±0.93	0.32
Bone [‡]	13.4±0.25	13.0±0.25	0.33
Tendon [‡]	1.0±0.06	0.8±0.06	0.05
Muscle:bone ratio	3.67±0.08	3.72±0.08	0.60

	Slaughter Group			Probability
	6 Weeks	12 Weeks	18 Weeks	
Number of animals	6	8	8	-
Half carcass weight, kg	21.7±0.86 ^a	25.7±0.74 ^b	28.7±0.74 ^c	<0.001
Dressing percentage [†]	55.7±0.62	55.6±0.53	55.9±0.53	0.93
Muscle [‡]	51.8±1.07 ^a	48.2±0.93 ^b	46.8±0.93 ^b	0.008
Fat [‡]	32.9±1.26 ^a	37.9±1.09 ^b	39.8±1.09 ^b	0.003
Bone [‡]	14.4±0.33 ^a	13.1±0.29 ^b	12.5±0.29 ^b	0.002
Tendon [‡]	0.8±0.08	0.9±0.07	0.9±0.07	0.90
Muscle:bone ratio	3.60±0.10	3.70±0.09	3.76±0.09	0.54

^f Warm carcass weight expressed as a percentage of live weight.[†] Expressed as a percentage of half carcass weight.

There were no significant treatment by slaughter group interactions for any of these traits.

Table V.8 Anatomical muscle group weights (expressed as a percentage of half carcass muscle weight) of control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		Probability
	Control	Exercised	
Number of animals	11	11	-
Total muscle weight, kg	12.35±0.33	12.57±0.33	0.64
1. Proximal pelvic limb	28.6±0.21	29.3±0.21	0.02
2. Distal pelvic limb	4.6±0.12	4.8±0.12	0.23
3. Around spinal column	16.3±0.21	15.4±0.21	<0.01
4. Abdominal wall	10.9±0.20	11.5±0.20	0.06
5. Proximal thoracic limb	13.0±0.13	13.2±0.13	0.44
6. Distal thoracic limb	2.7±0.06	2.8±0.06	0.15
7. Thorax to thoracic limb	8.6±0.15	8.3±0.15	0.12
8. Neck to thoracic limb	4.6±0.11	4.7±0.11	0.69
9. Neck and thorax	10.6±0.25	10.0±0.25	0.15

	Slaughter Group			Probability
	6 Weeks	12 Weeks	18 Weeks	
Number of animals	6	8	8	-
Total muscle weight, kg	11.29±0.45 ^a	12.42±0.39 ^b	13.39±0.39 ^c	0.01
1. Proximal pelvic limb	29.2±0.29	29.2±0.25	28.5±0.25	0.11
2. Distal pelvic limb	4.7±0.16	4.7±0.14	4.7±0.14	0.99
3. Around spinal column	15.9±0.29	15.9±0.25	15.7±0.25	0.83
4. Abdominal wall	10.6±0.27	11.3±0.23	11.5±0.23	0.09
5. Proximal thoracic limb	13.5±0.18	13.1±0.16	12.9±0.16	0.86
6. Distal thoracic limb	2.8±0.08	2.7±0.07	2.7±0.07	0.86
7. Thorax to thoracic limb	8.3±0.21	8.3±0.18	8.7±0.18	0.16
8. Neck to thoracic limb	4.6±0.15	4.5±0.13	4.8±0.13	0.24
9. Neck and thorax	10.3±0.34	10.3±0.30	10.4±0.30	0.97

There were no significant treatment by slaughter group interactions for any of these traits.

a,b,c means not followed by the same letter are significantly different at the 5% level of probability.

contrast to the changes between the two treatments, total muscle weight increased significantly and the distribution of muscle into anatomical muscle groups changed very little over the three slaughter groups. Muscles in the abdominal wall (Group 4) tended to increase as a proportion of total muscle over the three slaughter groups (10.6, 11.3, 11.5%; $P=0.09$).

Anatomical muscle groups which were significantly different between treatments and among slaughter groups at the 10% probability level were broken down into their individual muscle distributions (Table V.7). Exercised sheep had a significantly larger proportion of *biceps femoris* (5.55 vs. 5.17%) and *semimembranosus* (5.04 vs. 4.85%) than control sheep. Exercised sheep also tended to have a slightly larger proportion of the *gluteus medius* than control sheep (3.45 vs. 3.31%; $P=0.14$). In the abdominal muscles (Group 4), exercised sheep had a significantly larger proportion of *transversus abdominis* ($P=0.05$) and tended to have a significantly larger proportion of *obliquus externus abdominis* ($P=0.09$). The *longissimus dorsi* tended to be a lower proportion of total muscle weight in the exercised sheep than in the control sheep (9.48 vs. 10.00%). In addition, the *psoas minor*, *spinalis dorsi* and *longissimus cervicis* were all smaller proportions of total muscle weight in the exercised sheep than in the control sheep ($P=0.06$, $P=0.07$, $P=0.005$, respectively).

Muscle group 4 (the abdominal muscles) was the only group that showed significant differences among slaughter groups. The increased proportion of this group over the slaughter groups was mainly due to a significant increase in the proportions of *obliquus internus abdominis* ($P=0.02$) and *serratus dorsalis caudalis* ($P=0.04$). The *rectus abdominis* also tended to increase as a proportion of total muscle over the three slaughter groups (2.81, 2.78, 3.06%).

Bone distribution.

There were few changes in total bone weight or bone weight distribution. Exercised sheep had a slightly lower proportion of cervical vertebrae than control sheep (8.51 vs. 9.98%; $P=0.07$; Table V.8). Total bone weight increased significantly over the three slaughter groups ($P=0.01$) whereas the distribution of bone remained relatively constant over the three

Table V.7 Muscle distribution within selected anatomical muscle groups (individual muscle weights expressed as a percentage of half carcass muscle weight) of control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		Probability
	Control	Exercised	
Half carcass muscle weight, kg	12.3±0.33	12.6±0.33	0.64
Muscle Group 1 - Proximal Pelvic Limb			
1. Tensor fascia latae	1.04±0.039	1.12±0.039	0.18
2. Biceps femoris	5.17±0.079	5.55±0.079	0.004
3. Gluteus medius	3.31±0.065	3.45±0.065	0.14
4. Vastus lateralis	2.45±0.048	2.45±0.048	0.94
5. Gluteus accessorius	0.52±0.021	0.49±0.021	0.46
6. Gluteus profundus	0.31±0.015	0.31±0.015	0.99
7. Rectus femoris	2.27±0.037	2.30±0.037	0.60
8. Semitendinosus	1.91±0.047	1.98±0.047	0.26
9. Gracilis	0.95±0.051	0.88±0.051	0.34
10. Semimembranosus	4.85±0.075	5.04±0.075	0.09
11. Adductor femoris	1.96±0.051	1.95±0.051	0.81
12. Pectineus	0.53±0.014	0.52±0.014	0.62
13. Sartorius	0.20±0.013	0.19±0.013	0.39
14. Gemellus	0.05±0.004	0.05±0.004	0.49
15. Quadratus femoris	0.05±0.005	0.04±0.005	0.10
16. Obturator internus et externus	0.52±0.016	0.52±0.016	0.97
17. Vastus medialis	0.91±0.022	0.92±0.022	0.76
18. Vastus intermedius	0.82±0.021	0.77±0.021	0.13
19. Articularis genu	0.12±0.018	0.11±0.018	0.26
20. Iliacus	0.63±0.039	0.67±0.039	0.47

Continued

Table V.7 (cont'd.)

	Treatment		Probability
	Control	Exercised	
Muscle Group 3 - Spinal			
1. Psoas minor	0.58±0.019	0.52±0.019	0.06
2. Psoas major	1.52±0.043	1.51±0.043	0.83
3. Quadratus lumborum	0.31±0.015	0.33±0.015	0.40
4. Iliocostalis	0.50±0.014	0.48±0.014	0.32
5. Longissimus	9.99±0.228	9.48±0.228	0.13
6. Spinalis dorsi	1.62±0.103	1.33±0.103	0.07
7. Multifidus dorsi	1.60±0.075	1.56±0.075	0.75
8. Longissimus cervicis	0.21±0.013	0.15±0.013	0.005
Muscle Group 4 - Abdominal			
1. Cutaneous trunci	1.92±0.095	2.07±0.095	0.30
2. Serratus dorsalis caudalis	0.19±0.018	0.19±0.018	0.99
3. Obliquus externus abdominis	2.05±0.084	2.26±0.084	0.09
4. Retractor costae	0.20±0.019	0.21±0.019	0.62
5. Obliquus internus abdominis	1.42±0.045	1.38±0.045	0.49
6. Transversus abdominis	1.57±0.081	1.81±0.081	0.05
7. Rectus abdominis	2.91±0.091	2.88±0.091	0.83
8. Diaphragm	0.64±0.025	0.67±0.025	0.29

Cont Inued

Table V.7 (Cont'd.)

	6 Weeks	Slaughter Group 12 Weeks	18 Weeks	Probability
Half carcass muscle weight, kg	11.3±0.45 ^a	12.4±0.39 ^b	13.4±0.39 ^c	0.01
Muscle Group 4 - Abdominal				
1. Cutaneous trunci	1.91±0.128	2.11±0.111	1.95±0.111	0.43
2. Serratus dorsalis caudalis	0.15±0.024 ^a	0.18±0.021 ^{a,b}	0.24±0.021 ^b	0.04
3. Obliquus externus abdominis	2.08±0.114	2.14±0.099	2.23±0.099	0.61
4. Retractor costae	0.20±0.026	0.19±0.022	0.23±0.022	0.44
5. Obliquus internus abdominis	1.25±0.061 ^a	1.42±0.053 ^b	1.49±0.053	0.02
6. Transversus abdominis	1.63±0.109	1.80±0.095	1.63±0.095	0.38
7. Rectus abdominis	2.81±0.123	2.78±0.107	3.06±0.107	0.16
8. Diaphragm	0.63±0.034	0.69±0.029	0.64±0.029	0.40

^{a,b} Means not followed by the same letter are significantly different at the 5% level of probability.

Table V.8 Bone distribution (individual bone weights expressed as a percentage of half carcass bone weight) of control and endurance-exercised sheep split by treatment.

	Treatment		Probability
	Control	Exercised	
Number of animals	11	11	—
Total bone weight, kg	3.40±0.06	3.38±0.06	0.81
Axial Skeleton			
Atlas	52.3±0.55	53.2±0.55	0.32
Cervical vertebrae	1.87±0.07	1.84±0.07	0.77
Thoracic vertebrae	9.98±0.53	8.51±0.53	0.07
Lumbar vertebrae	12.05±0.47	12.00±0.47	0.94
Costae	7.85±0.31	7.78±0.31	0.88
Sternum and costal cartilage	13.37±0.29	13.51±0.29	0.74
	8.09±0.44	8.70±0.44	0.34
Appendicular Skeleton			
Scapula	47.6±0.55	46.8±0.55	0.28
Humerus	4.89±0.08	5.05±0.08	0.17
Radius	6.43±0.08	6.50±0.08	0.55
Carpus	5.35±0.08	5.50±0.08	0.22
Os Coxae	0.93±0.13	1.14±0.13	0.27
Femur	9.47±0.27	9.49±0.27	0.96
Tibia	8.38±0.10	8.37±0.10	0.93
Patella	6.85±0.15	6.94±0.15	0.69
Tarsus	0.58±0.011	0.60±0.011	0.28
	3.90±0.06	3.38±0.06	0.81

There were no significant treatment by slaughter group interactions for any of these traits.

slaughter groups (Table V.9).

Fat distribution

There were no significant differences in total fat weight, absolute fat depot weights, or the proportion of fat in the depots between exercised and control sheep (Table V.10). Among slaughter groups, total fat increased with time on trial (7.2, 9.8, 11.4 kg; $P < 0.01$). Similarly, absolute weights of the fat depots (subcutaneous, SCF; intermuscular, IMF; and perirenal plus channel fat, PRCF) increased significantly over the three slaughter groups. As a proportion of total fat, the IMF depot decreased and the PRCF depot increased over the three slaughter groups ($P = 0.01$, $P = 0.03$). There was a significant treatment by slaughter group interaction effect for the SCF and PRCF depots expressed as a percentage of total fat (Figure V.8).

Muscle fiber diameters and sarcomere lengths

There was no indication of a bimodal fiber diameter distribution for any of the five muscles studied. Muscles from exercised sheep all had larger mean fiber diameters than muscles from control sheep (Table V.11); however, the large standard errors of the means resulted in very few significant differences. VL muscles from exercised animals tended to have larger diameters than VL muscles from control sheep (51.2 vs. 55.5 μm ; $P = 0.08$). The standard deviation of the mean tended to be larger in ST and G muscles from exercised sheep when compared to their control counterparts. In addition, the distribution of the ST fiber diameter means measured in the exercised sheep was significantly skewed towards the larger diameters ($P = 0.03$). With the exception of the ST, there appeared to be a trend toward larger fiber diameters in the 18 week slaughter group than in the 6 week slaughter group in the SM ($P = 0.34$), VL ($P = 0.16$), PM ($P < 0.01$) and G ($P = 0.06$). The standard deviation of the mean for the PM and G was significantly larger in the 18 week slaughter group than in the 6 or 12 week slaughter groups ($P < 0.01$ in both muscles).

Table V.9 Bone distribution (individual bone weights expressed as a percentage of half carcass bone weight) of control and endurance-exercised sheep split by slaughter group.

	Slaughter Group			Probability
	6 Weeks	12 Weeks	18 Weeks	
Number of animals	6	8	8	-
Total bone weight, kg	3.19±0.08 ^a	3.35±0.07 ^{a,b}	3.56±0.07 ^b	0.01
Axial Skeleton				
Atlas	53.2±0.75	52.5±0.65	52.7±0.65	0.81
Cervical vertebrae	1.8±0.10	2.0±0.08	1.75±0.08	0.12
Thoracic vertebrae	9.8±0.72	8.4±0.62	9.6±0.62	0.27
Lumbar vertebrae	11.4±0.63	12.59±0.55	11.90±0.55	0.39
Costae	8.2±0.42	7.60±0.36	7.73±0.36	0.53
Sternum and costal cartilage	12.95±0.39	13.28±0.34	13.97±0.34	0.15
	8.93±0.60	8.66±0.52	7.74±0.29	0.29
Appendicular Skeleton				
Scapula	46.8±0.75	47.5±0.65	47.2±0.65	0.81
Humerus	4.80±0.10	4.96±0.09	5.11±0.09	0.11
Radioulna	6.48±0.11	6.53±0.10	6.39±0.10	0.61
Carpus	5.35±0.11	5.49±0.10	5.41±0.10	0.63
Os coxae	1.07±0.18	1.17±0.15	0.86±0.15	0.36
Femur	9.91±0.37	9.13±0.32	9.51±0.32	0.31
Tibia	8.31±0.14	8.46±0.12	8.35±0.12	0.71
Patella	6.60±0.21	7.05±0.18	6.95±0.18	0.29
Tarsus	0.58±0.015	0.62±0.013	0.57±0.013	0.07
	3.73±0.19	4.06±0.17	4.11±0.17	0.31

^{a,b} Means not followed by the same letter are significantly different at the 5% level of probability. There were no significant treatment by slaughter group interactions for any of these traits.

Table V.10 Fat distribution (absolute fat depot weights and fat depot weights as a percentage of half carcass fat weight) of control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		
	Control	Exercised	Probability
Number of animals	11	11	—
Total fat, kg	9.3±0.40	10.0±0.40	0.25
Absolute weight			
Subcutaneous fat, kg	4.96±0.27	5.43±0.27	0.23
Intermuscular fat, kg	3.23±0.18	3.37±0.18	0.61
Perirenal plus channel fat, kg	2.45±0.18	2.41±0.18	0.89
Percentage of total fat			
Subcutaneous, %	46.2±1.38	48.8±1.38	0.21
Intermuscular, %	30.9±0.99	30.2±0.99	0.65
Perirenal plus channel fat, %	22.8±1.07	20.9±1.07	0.23
Slaughter Group			
	6 Weeks	12 Weeks	18 Weeks
Number of animals	6	8	8
Total fat, kg	7.2±0.55 ^a	9.8±0.47 ^b	11.5±0.47 ^c
Absolute weight			
Subcutaneous fat, kg	3.55±0.36 ^a	5.49±0.32 ^b	6.05±0.32 ^b
Intermuscular fat, kg	2.73±0.25 ^a	3.10±0.21 ^a	3.94±0.21 ^b
Perirenal plus channel fat, kg	1.56±0.25 ^a	2.19±0.22 ^a	3.32±0.22 ^b
Percentage of total fat			
Subcutaneous, %	45.8±1.87	50.6±1.62	45.7±1.62
Intermuscular, %	34.4±1.34 ^a	28.8±1.16 ^b	29.5±1.16 ^b
Perirenal plus channel fat, %	19.7±1.45 ^a	20.6±1.25 ^a	24.8±1.25 ^b
			0.09
			0.01
			0.03

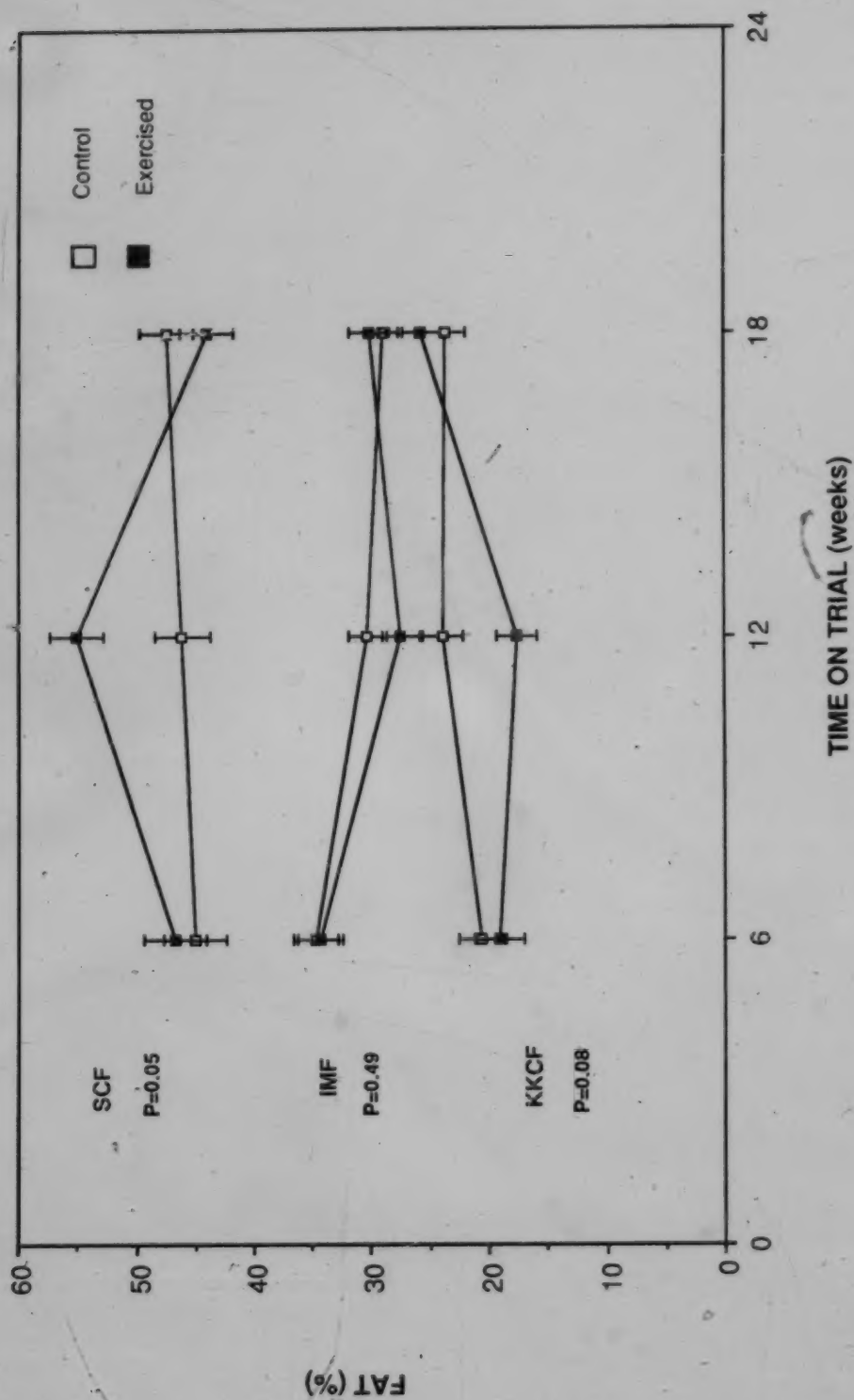
a, b, c Means not followed by the same letter are significantly different at the 5% level of probability.

Table V.11 Muscle fiber diameters (not adjusted to a constant sarcomere length) of control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		Probability
	Control	Exercised	
Number of animals	11	11	-
Diameters, micrometers			
Semimembranosus	50.6±1.4	52.5±1.4	0.37
Vastus lateralis	51.2±1.7	55.5±1.7	0.08
Semitendinosus	43.5±0.9	44.0±0.9	0.73
Psoas major	34.8±0.9	36.2±0.9	0.29
Gastrocnemius	46.9±1.9	48.2±1.9	0.64
Slaughter Group			
	6 Weeks	12 Weeks	18 Weeks
Probability			
Number of animals	6	8	8
Diameters, micrometers			
Semimembranosus	49.3±1.9	51.4±1.7	53.3±1.7
Vastus lateralis	51.4±2.2	51.7±1.9	56.5±1.9
Semitendinosus	43.8±1.2	43.9±1.1	43.6±1.1
Psoas major	32.7±1.2 ^a	32.8±1.1 ^a	40.3±1.1 ^b
Gastrocnemius	43.0±2.6	46.6±2.3	51.9±2.3
Probability			
			0.34
			0.16
			0.97
			<0.01
			0.06

a, b Means not followed by the same letter are significantly different at the 5% level of probability.

Figure V.8 Treatment by slaughter group interactions for fat depot weights (expressed as a proportion of half carcass fat weight)



Sarcomere lengths were shorter in the exercised sheep in all five muscles ($P=0.04$; Figure V.9). Since a shorter sarcomere length results in a larger muscle fiber diameter, the muscle fiber diameters were adjusted to the average sarcomere length for each muscle (SM 2.19, VL 2.38, ST 2.84, PM 2.84, G 2.90 μm). There was no consistent pattern to changes in sarcomere length over the three slaughter groups (Figure V.10). The SM was the only muscle for which there was a significant relationship between sarcomere length and carcass weight; as carcass weight increased, sarcomere length increased.

The adjusted muscle fiber diameter means are presented in Table V.12. The difference in the mean fiber diameter between the exercised and control sheep was reduced for all the muscles, resulting in higher probability levels. The probability level increased in the VL from 0.08 to 0.14 and increased in the PM from 0.29 to 0.89. The tendency towards significantly different standard deviations in the ST and G from control and exercised sheep was reduced. However, the ST from exercised sheep still had a distribution of means significantly skewed towards the larger diameters ($P=0.03$). The same trends still existed over the three slaughter groups. The 18 week slaughter group had larger fiber diameters ($P<0.01$) and larger standard deviations ($P<0.01$) than both the 6 and 12 week slaughter groups in the PM (Figure V.11). In the G, the standard deviations increased over the three slaughter groups ($P=0.10$, $P<0.01$, respectively). There was a significant ($P=0.07$) adjustment by treatment interaction, indicating that the adjustment to a constant sarcomere length was non-linear between the two treatments.

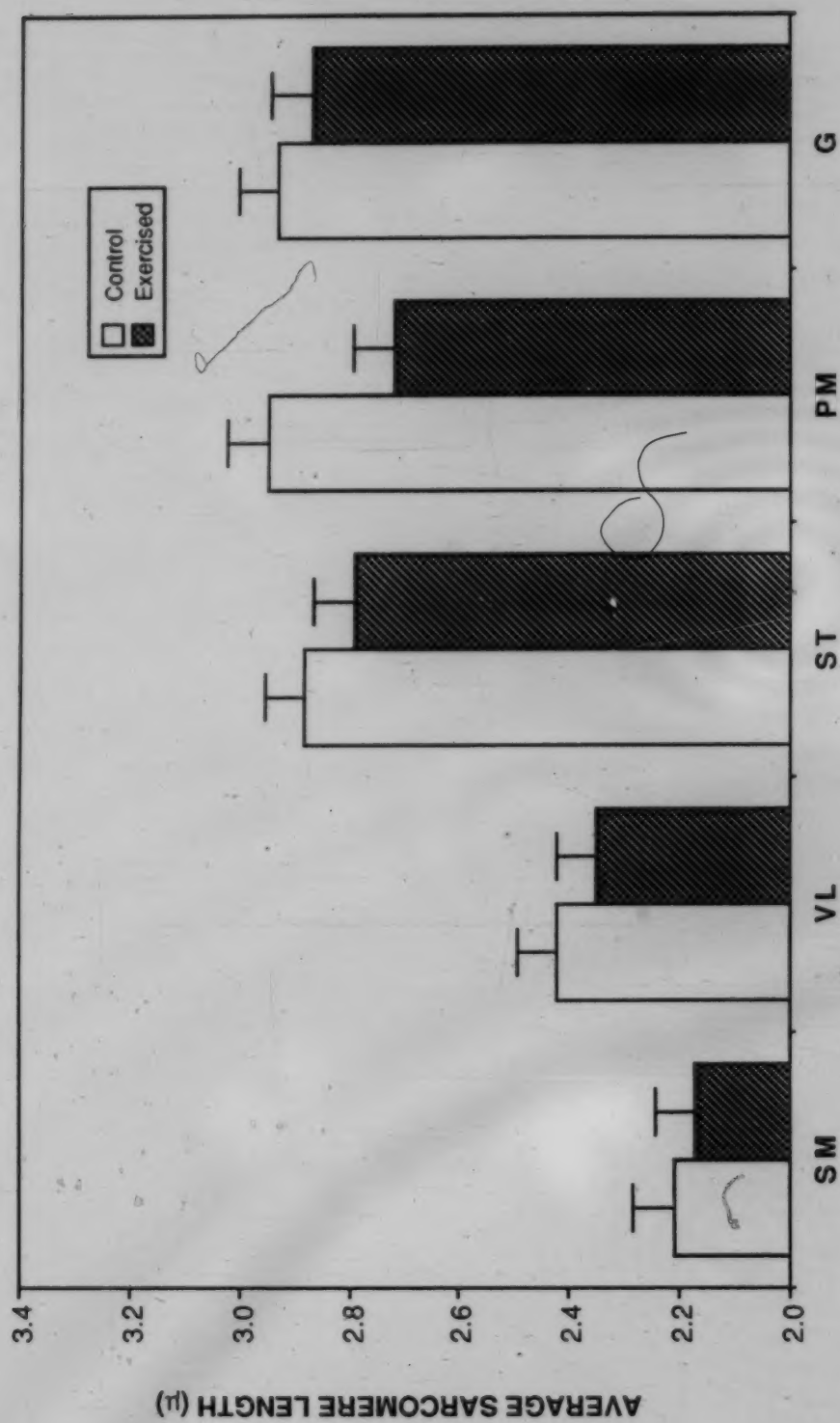
The SM and VL had the largest muscle fiber diameters and the shortest sarcomere lengths (Figure V.12). The ST, PM and G had the longest sarcomere lengths and were not significantly different from one another (2.84, 2.84, 2.90 μm , respectively). The PM had the smallest fiber diameters (35.5 μm) followed by the ST (43.8 μm) and then the G (47.6 μm). On a volumetric basis ($V = \text{SL} \times \pi/4 \times D^2$; SL is sarcomere length, D is fiber diameter), the VL had the largest myofibrillar volume, followed in declining order by the G, SM, ST and PM.

Table V.12 Muscle fiber diameters (adjusted to a constant sarcomere length for each muscle) of control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		
	Control	Exercised	Probability
Number of animals	11	11	-
Diameters, micrometers			
<i>Semimembranosus</i>	50.7±1.5	52.3±1.5	0.48
<i>Vastus lateralis</i>	51.6±1.6	55.0±1.6	0.14
<i>Semitendinosus</i>	43.8±0.9	43.6±0.9	0.88
<i>Psoas major</i>	35.3±1.2	35.5±1.2	0.89
<i>Gastrocnemius</i>	47.0±1.9	47.9±1.9	0.74
Slaughter Group			
	6 Weeks	12 Weeks	18 Weeks
Probability			
Number of animals	6	8	8
Diameters, micrometers			
<i>Semimembranosus</i>	48.3±2.0	51.5±1.7	54.0±1.7
<i>Vastus lateralis</i>	51.9±2.1	51.8±1.9	55.9±1.9
<i>Semitendinosus</i>	43.9±2.1	44.1±1.9	43.3±1.9
<i>Psoas major</i>	33.1±1.6 ^a	33.1±1.4 ^a	39.4±1.4 ^b
<i>Gastrocnemius</i>	42.5±2.6	48.5±2.2	50.1±2.2
Probability			0.12 0.24 0.89 <0.01 0.10

^{a,b}Means not followed by the same letter are significantly different at the 5% level of probability.

Figure V.9 Average sarcomere lengths of the *semimembranosus*, *vastus lateralis*, *semitendinosus*, *psoas major* and *gastrocnemius* muscles from control and endurance-exercised sheep split by treatment



Repeated measures analysis: Control > Exercised, $P=0.04$

Figure V.11 Average muscle fiber diameters (adjusted to a constant sarcomere length) of the *semimembranosus*, *vastus lateralis*, *semitendinosus*, *psaos major* and *gastrocnemius* muscles from control and endurance-exercised sheep split by slaughter group

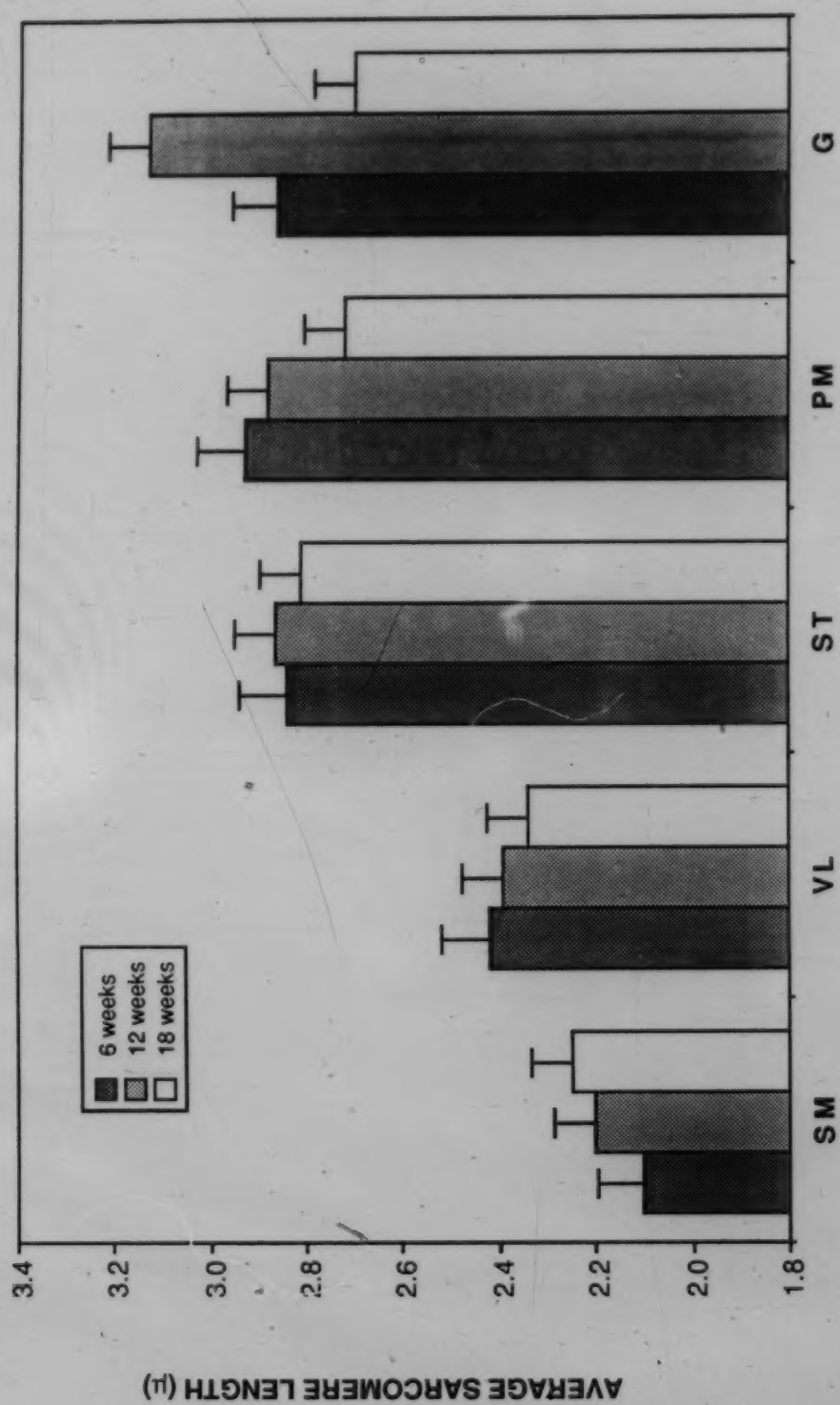


Figure V.10 Average sarcomere lengths of the *semimembranosus*, *vastus lateralis*, *semitendinosus*, *psoas major* and *gastrocnemius* muscles from control and endurance-exercised sheep split by slaughter group

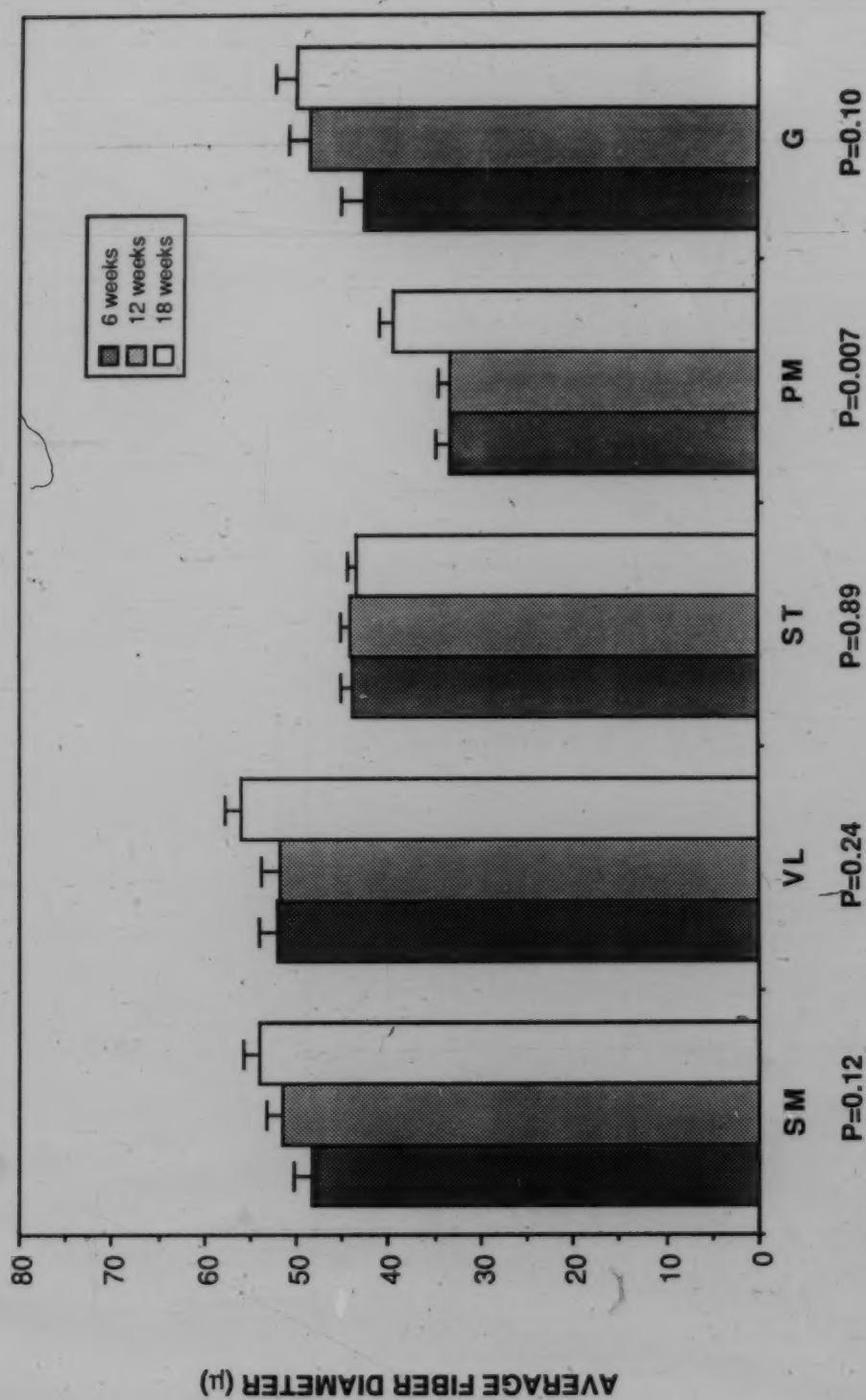
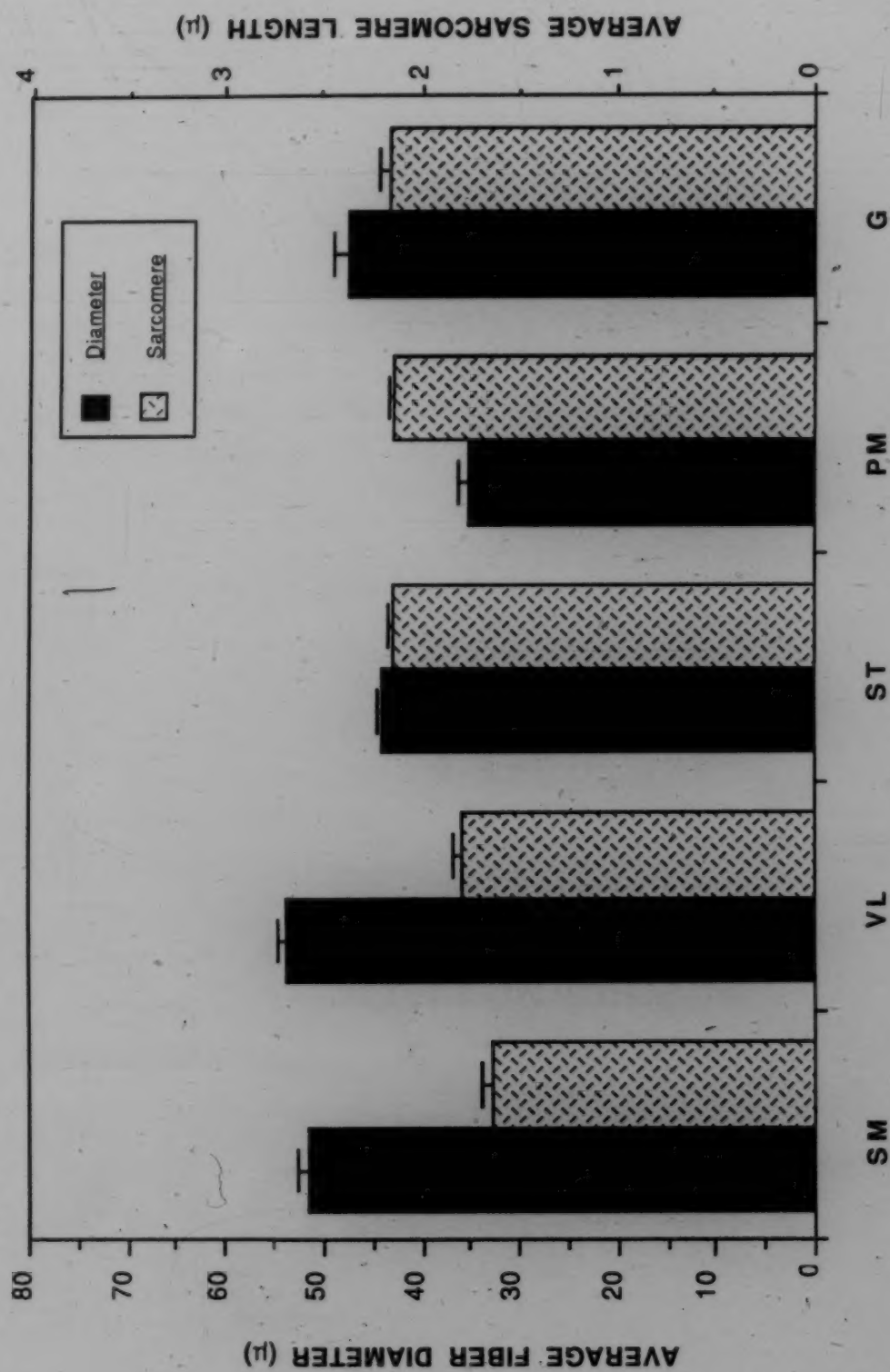


Figure V.12 Average muscle fiber diameters and sarcomere lengths for the *semimembranosus*, *vastus lateralis*, *semitendinosus*, *psoas major* and *gastrocnemius* muscles



Post-mortem pH and temperature decline

Post-mortem temperature declines of the five muscles (SM, VL, ST, PM and G) are shown on Figure V.13. It was necessary to remove the layer of internal body fat covering the PM and to reflect the *biceps femoris* overlying the G in order to obtain pH and temperature readings. Not surprisingly, the G and PM then had the most rapid rate of temperature decline. Similarly, the SM and VL, superficial muscles which were covered by a thick layer of subcutaneous fat, had intermediate rates of temperature decline. The ST, anatomically the deepest muscle of the five measured, had the lowest rate of temperature decline. There were no treatment or slaughter group differences in rates of temperature decline for any of the five muscles.

There were marked differences in post-mortem pH decline among the five muscles (Figure V.14). Ranked in order from the highest to the lowest rate of pH decline, they were: PM>ST>SM>VL>G. The rate of pH decline was not related to the final 24 hour pH. At 24 hours post-mortem, the G had the highest pH, followed by the ST, PM/VL, and SM.

Treatment differences in post-mortem pH decline are shown for individual muscles on Figure 15 (a-e). The ST from exercised sheep had a significantly slower pH decline post-mortem than the ST from control sheep ($P<0.01$). The SM and PM from exercised animals also tended to have slower pH declines post-mortem than their counterparts from control animals. Slaughter group differences in post-mortem pH decline are shown for individual muscles on Figure 16 (a-e). The 6 week slaughter group had a significantly higher rate of post-mortem pH decline than either the 12 or 18 week slaughter groups for four of the five muscles (SM, VL, PM and G).

Muscle fiber types

Exercised sheep had significantly higher percentages of slow-contracting fibers (alkaline myosin ATPase light-staining fibers) in the SM, VL, ST and G when compared with control sheep (Figure V.17). The percentage of slow-contracting fibers in the PM was significantly larger in the 18 week slaughter group than in either the 6 or 12 week slaughter

Figure V.13 Post-mortem temperature decline of the *semimembranosus*, *vastus lateralis*,
semitendinosus, *psoas major* and *gastrocnemius* muscles

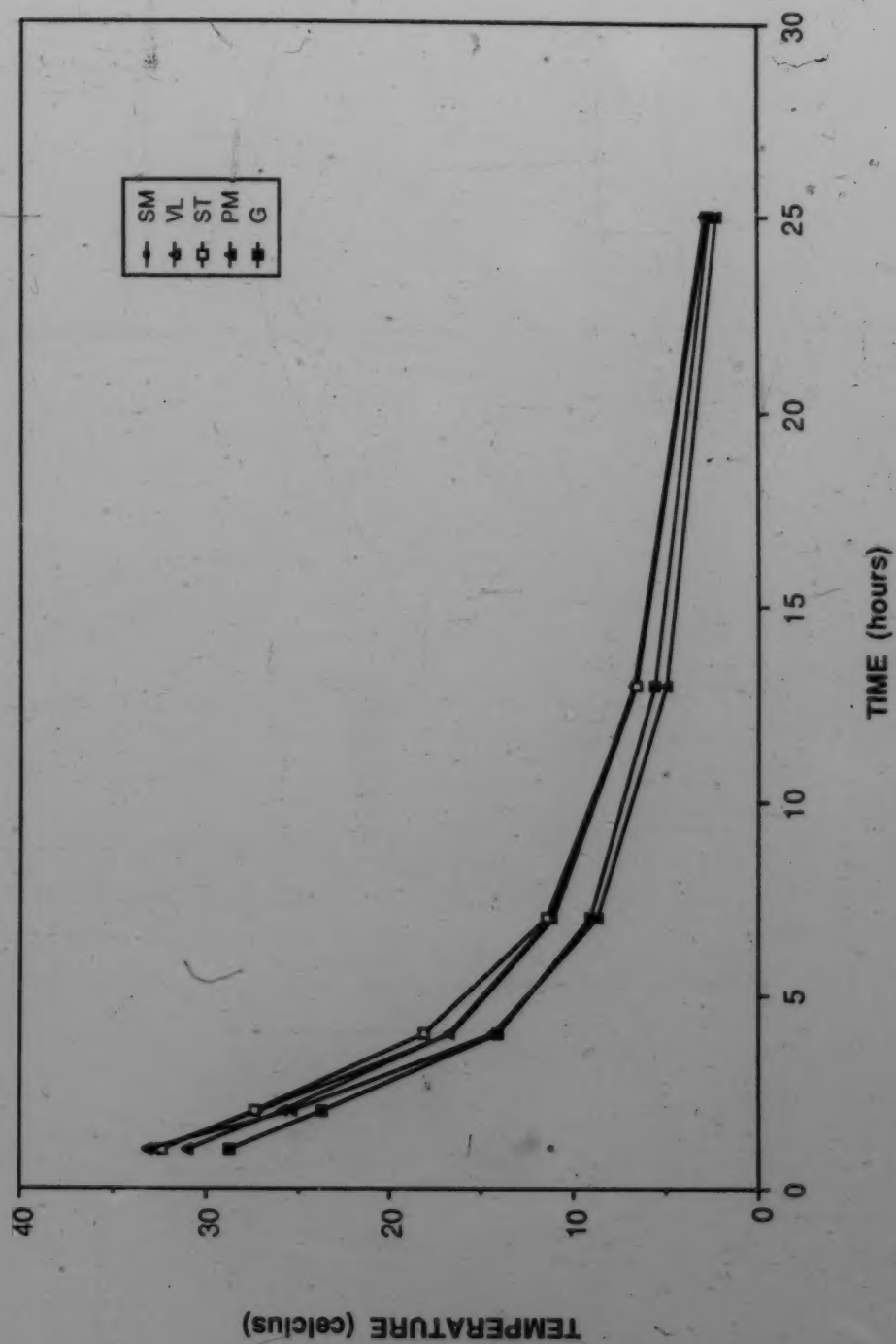


Figure V.14 Post-mortem pH decline of the *semimembranosus*, *vastus lateralis*, *semitendinosus*, *psoas major* and *gastrocnemius* muscles

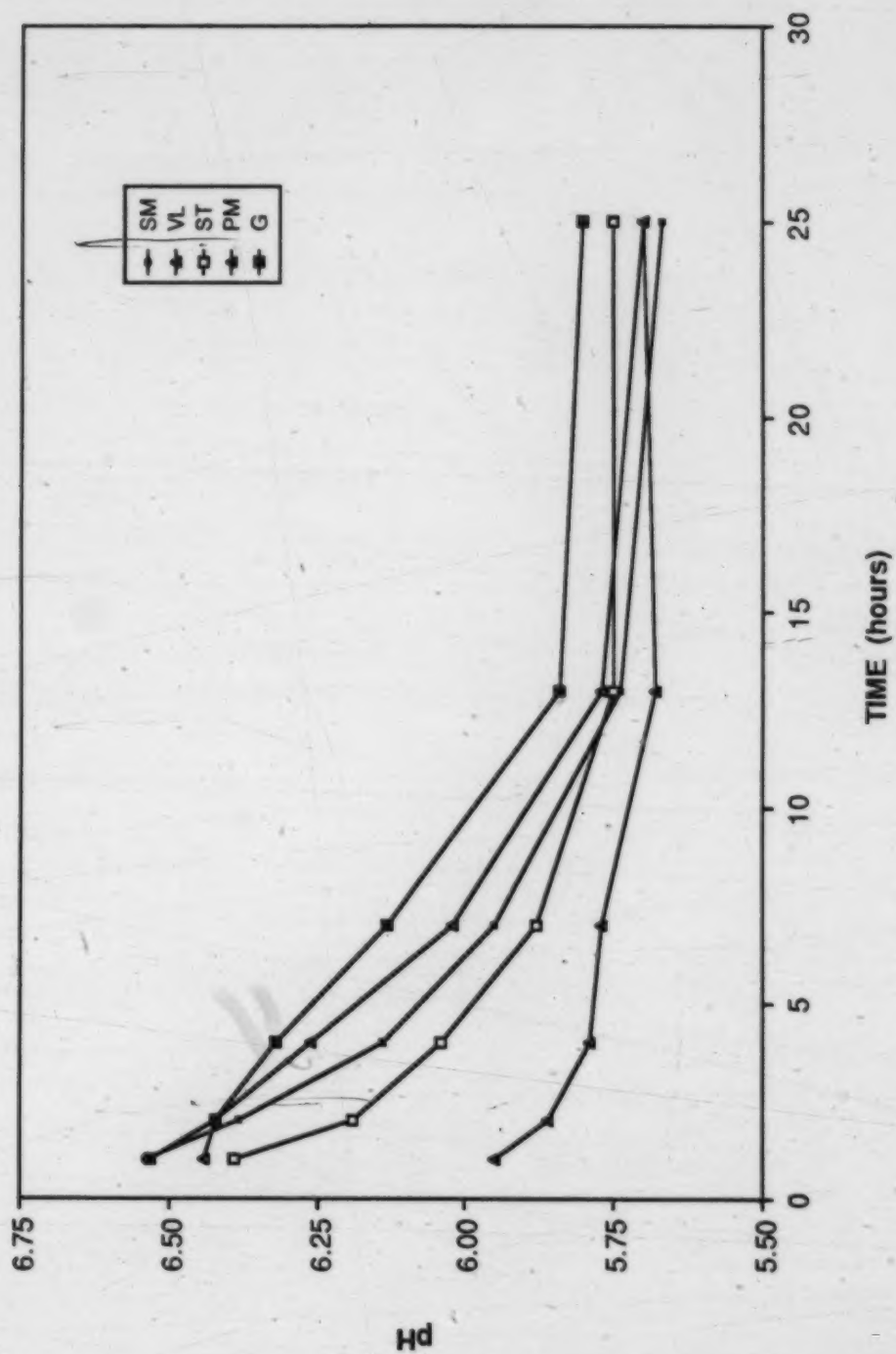


Figure V.15 Treatment differences in post-mortem pH decline of the a) *semimembranosus*, b) *vastus lateralis*, c) *semitendinosus*, d) *psoas major*, e) *gastrocnemius* muscles

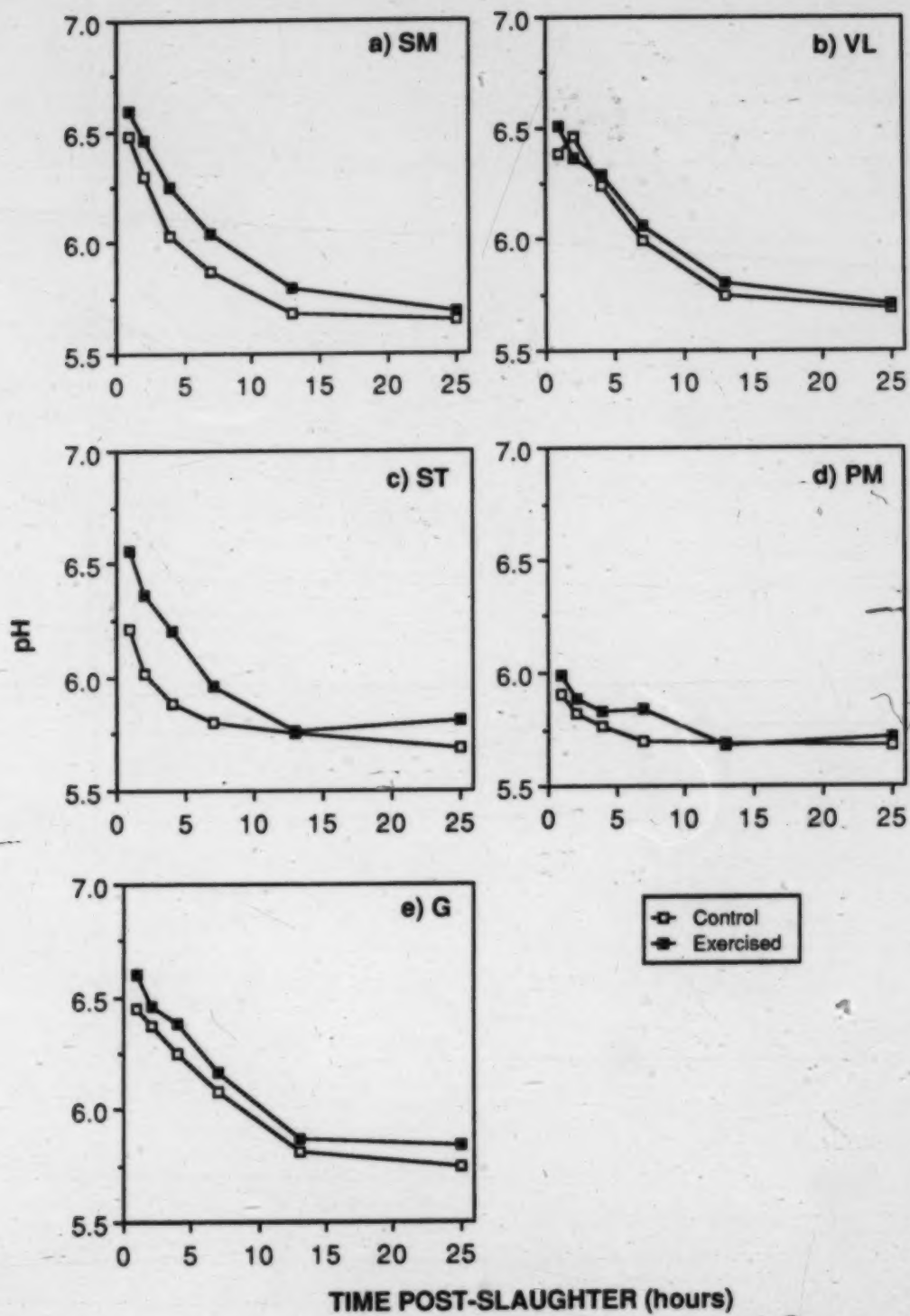


Figure V.16 Slaughter group differences in post-mortem pH decline of the a) *semimembranosus*, b) *vastus lateralis*, c) *semitendinosus*, d) *psoas major*, e) *gastrocnemius* muscles

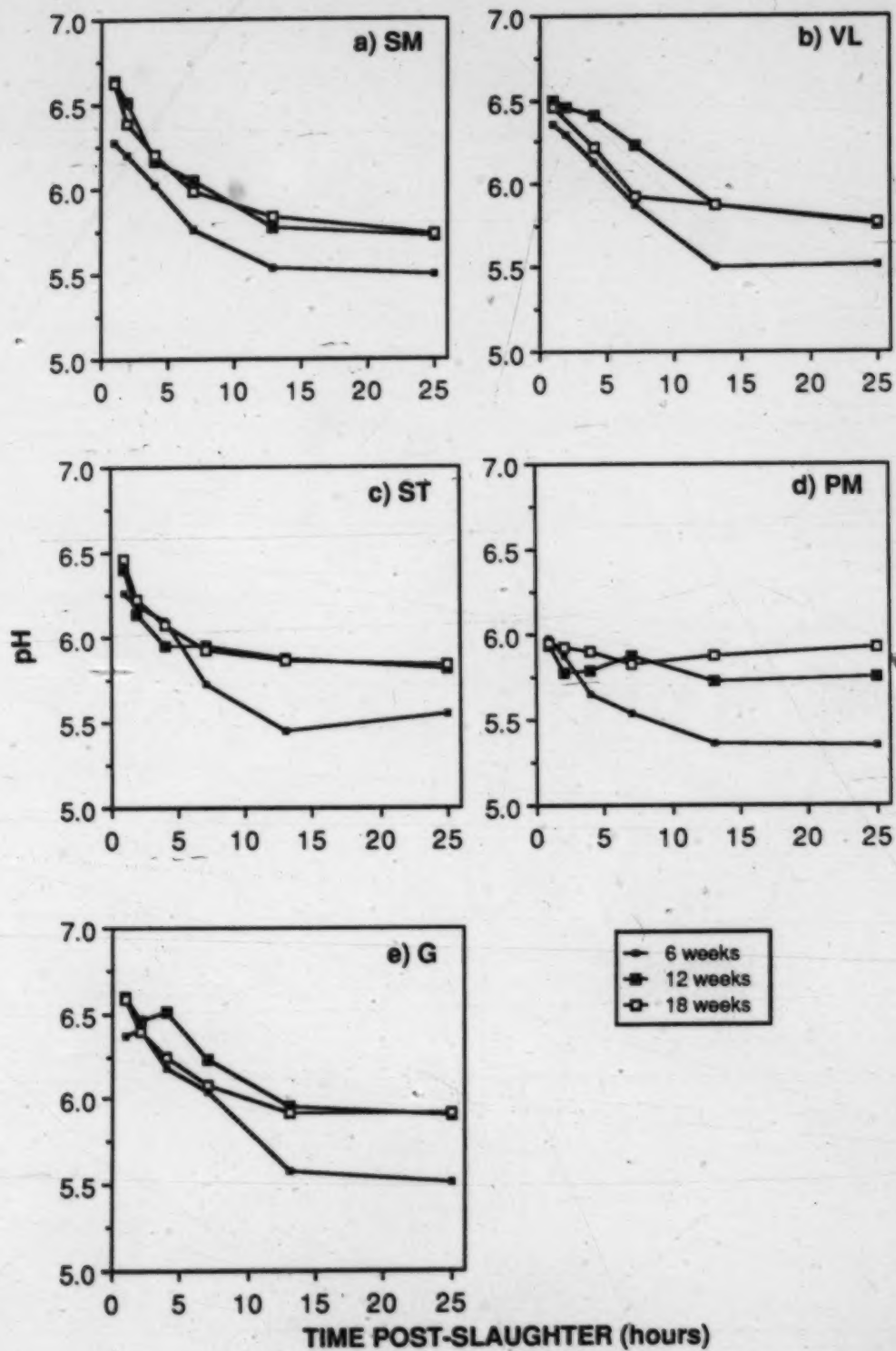
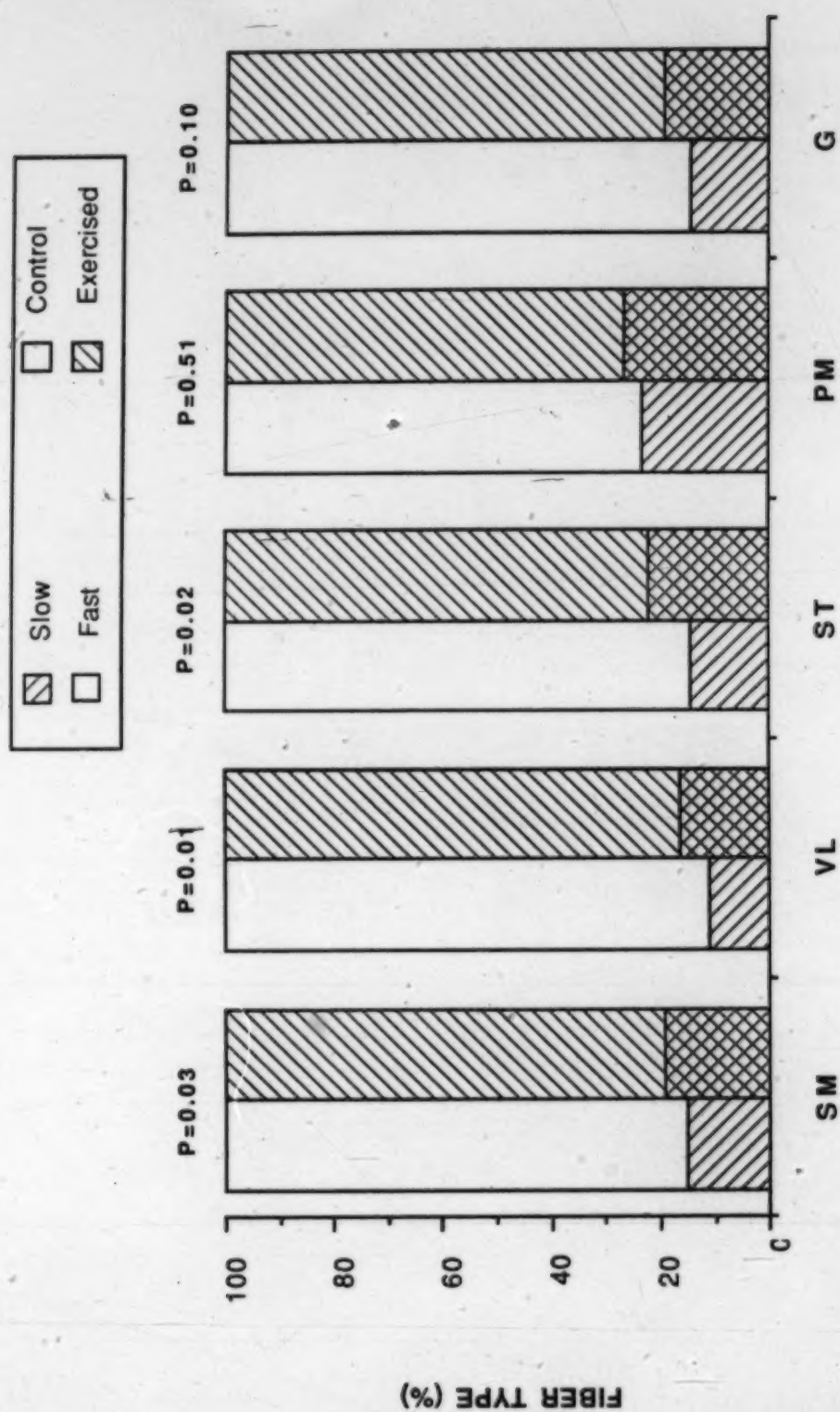


Figure V.17 Treatment differences in the percentage of slow fibers (classified using the myosin ATPase stain)



groups (Table V.13).

There were no significant treatment differences in the percentage of glycolytic or oxidative fibers for any of the muscles using the NADH dehydrogenase stain (Table V.14). However, the percentage of oxidative-glycolytic fibers was significantly smaller in the ST ($P < 0.01$) and G ($P = 0.02$) from exercised sheep when compared to the ST and G from control sheep (Figure V.18). There was a significant difference among slaughter groups for both the oxidative-glycolytic ($P = 0.02$) and glycolytic ($P = 0.04$) fibers in the SM (Table V.15). There tended to be a higher percentage of oxidative-glycolytic fibers in the 6 week slaughter group as compared to the 12 and 18 week slaughter groups in the ST ($P = 0.10$). In the G, there was a slightly higher percentage ($P = 0.09$) of oxidative fibers in muscles from the 18 week slaughter group compared to muscles from the 6 and 12 week slaughter groups.

There were four fiber types exhibited in each muscle when the two stains were combined: slow oxidative (SO), fast oxidative (FO), fast oxidative-glycolytic (FOG) and fast glycolytic (FG) (Figure V.19). The PM tended to have the largest percentage of oxidative fibers whereas the ST tended to have the lowest proportion of oxidative fibers. The VL and G tended to have the highest proportion of glycolytic fibers whereas the PM had the lowest percentage of glycolytic fibers.

Meat quality

Exercised sheep had more tender *semimembranosus* and *vastus lateralis* than control sheep (Figure V.20). Warner Bratzler shear values were significantly lower in both the SM and VL from exercised sheep as compared to control sheep (5.80, 5.00 vs. 7.04, 5.71 kg). Kramer Press ground values were also significantly lower in the VL from exercised sheep (3.79 vs. 4.32 kg.g⁻¹; $P = 0.01$). There were no differences between exercised and control sheep in ultimate pH for either the SM or VL (Table V.16). Among slaughter groups, the Kramer Press ground values increased significantly with time on trial (3.65 vs. 4.13 vs. 4.28 kg.g⁻¹) in the VL. Overall mechanical tenderness values were lower in the VL, indicating that this muscle was more tender than the SM.

Table V. 13 Percentage slow fibers (classified using the alkaline myosin ATPase stain) of control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		Probability
	Control	Exercised	
Number of animals	11	11	-
Myosin ATPase 'slow' fibers, %			
<i>Semimembranosus</i>	14.7±1.18	19.0±1.12	0.03
<i>Vastus lateralis</i>	11.7±1.11	17.0±1.11	<0.01
<i>Semitendinosus</i>	14.1±1.94	21.6±2.05	0.02
<i>Psoas major</i>	23.3±2.74	25.9±2.62	0.51
<i>Gastrocnemius</i>	15.0±1.91	19.7±1.91	0.10
Slaughter Group			
	6 Weeks	12 Weeks	18 Weeks
Probability			
Number of animals	6	8	8
Myosin ATPase 'slow' fibers, %			
<i>Semimembranosus</i>	18.4±1.76	15.2±1.25	17.0±1.33
<i>Vastus lateralis</i>	15.5±1.50	13.7±1.30	13.8±1.30
<i>Semitendinosus</i>	21.1±2.74	16.9±2.51	15.5±2.17
<i>Psoas major</i>	19.0±3.88 ^a	20.3±3.07 ^a	34.5±3.07 ^b
<i>Gastrocnemius</i>	17.3±2.59	16.6±2.24	18.2±2.24

a, b Means not followed by the same letter are significantly different at the 5% level of probability.

Table V. 14 Percentage oxidative, oxidative-glycolytic and glycolytic fiber types (classified using the NADH dehydrogenase stain) of control and endurance-exercised sheep split by treatment.

Number of animals	Treatment		Probability
	Control	Exercised	
<i>Semimembranosus</i>	11	11	-
Oxidative	33.8±2.07	30.9±1.76	0.32
Oxidative-glycolytic	45.1±2.18	46.6±1.86	0.62
Glycolytic	21.1±2.43	22.5±2.07	0.67
<i>Vastus lateralis</i>			
Oxidative	30.1±1.68	32.0±1.60	0.42
Oxidative-glycolytic	43.2±2.91	39.9±2.78	0.42
Glycolytic	26.6±3.30	28.1±3.15	0.76
<i>Semitendinosus</i>			
Oxidative	20.7±2.23	24.9±2.23	0.20
Oxidative-glycolytic	57.5±1.89	48.9±1.89	<0.01
Glycolytic	21.8±2.51	26.2±2.51	0.23
<i>Psoas major</i>			
Oxidative	47.0±3.08	44.6±3.24	0.61
Oxidative-glycolytic	33.0±2.75	33.6±2.88	0.89
Glycolytic	20.0±2.23	21.8±2.34	0.59
<i>Gastrocnemius</i>			
Oxidative	29.0±2.40	32.8±2.40	0.29
Oxidative-glycolytic	47.9±1.99	40.5±1.99	0.02
Glycolytic	23.1±2.53	26.7±2.53	0.33

Table V. 15 Percentage oxidative, oxidative-glycolytic and glycolytic fiber types (classified using the NADH dehydrogenase stain) of control and endurance-exercised sheep split by slaughter group.

	Slaughter Group			Probability
	6 Weeks	12 Weeks	18 Weeks	
Number of animals	6	8	8	-
<i>Semimembranosus</i>				
Oxidative	31.9±2.62	31.9±2.21 ^b	32.7±2.21	0.95
Oxidative-glycolytic	51.7±2.75 ^a	40.1±2.33 ^b	46.0±2.33 ^{a,b}	0.02
Glycolytic	16.4±3.08 ^a	28.0±2.60 ^b	21.3±2.60 ^{a,b}	0.04
<i>Vastus lateralis</i>				
Oxidative	31.9±2.17	29.7±2.01	31.7±1.88	0.70
Oxidative-glycolytic	44.5±3.76	37.2±3.48	42.9±3.26	0.34
Glycolytic	23.6±4.26	33.1±3.94	25.4±3.69	0.24
<i>Semitendinosus</i>				
Oxidative	21.3±3.02	21.4±2.61	25.7±2.61	0.45
Oxidative-glycolytic	57.8±2.56	51.0±2.22	50.8±2.22	0.10
Glycolytic	20.9±3.39	27.5±2.94	23.5±2.94	0.34
<i>Psoas major</i>				
Oxidative	46.4±4.58	41.0±3.62	50.0±3.62	0.24
Oxidative-glycolytic	33.2±4.07	35.5±3.22	31.1±3.22	0.54
Glycolytic	20.4±3.31	23.5±2.62	18.8±2.62	0.46
<i>Gastrocnemius</i>				
Oxidative	28.7±3.24	27.6±2.81	36.4±2.81	0.09
Oxidative-glycolytic	47.8±2.69	42.5±2.33	42.4±2.33	0.26
Glycolytic	23.4±3.43	30.0±2.97	21.2±2.97	0.13

a,b Means not followed by the same letter are significantly different at the 5% level of probability.

Table V. 16 Mechanical tenderness tests and ultimate pH of the semimembranosus¹ and vastus lateralis muscles from control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		
	Control	Exercised	Probability
Number of animals	11	11	-
<i>Semimembranosus</i>			
Warner Bratzler Shear, kg	7.04±0.32	5.80±0.32	0.01
Kramer Press - Ground, kg.g ⁻¹	5.02±0.24	4.62±0.24	0.26
Ultimate pH	5.56±0.034	5.53±0.034	0.50
<i>Vastus lateralis</i>			
Warner Bratzler Shear, kg	5.71±0.21	5.00±0.21	0.03
Kramer Press - Ground, kg.g ⁻¹	4.32±0.14	3.79±0.14	0.01
Ultimate pH	5.80±0.023	5.76±0.023	0.32
Slaughter Group			
	6 Weeks	12 Weeks	18 Weeks
Probability			
Number of animals	6	8	8
<i>Semimembranosus</i>			
Warner Bratzler Shear, kg	6.79±0.43	6.11±0.39	6.45±0.39
Kramer Press - Ground, kg.g ⁻¹	4.77±0.32	4.88±0.28	4.81±0.28
Ultimate pH	5.62±0.046	5.49±0.040	5.55±0.040
<i>Vastus lateralis</i>			
Warner Bratzler Shear, kg	5.45±0.28	5.41±0.24	5.22±0.24
Kramer Press - Ground, kg.g ⁻¹	3.65±0.18 ^a	4.13±0.16 ^b	4.28±0.16 ^b
Ultimate pH	5.76±0.031	5.80±0.026	5.77±0.026

^{a,b}Means not followed by the same letter are significantly different at the 5% level of probability. There were no significant treatment by slaughter group interactions for any of these traits.

Figure V.18 Treatment differences in the percentage of oxidative-glycolytic fibers (classified using the NADH dehydrogenase stain)

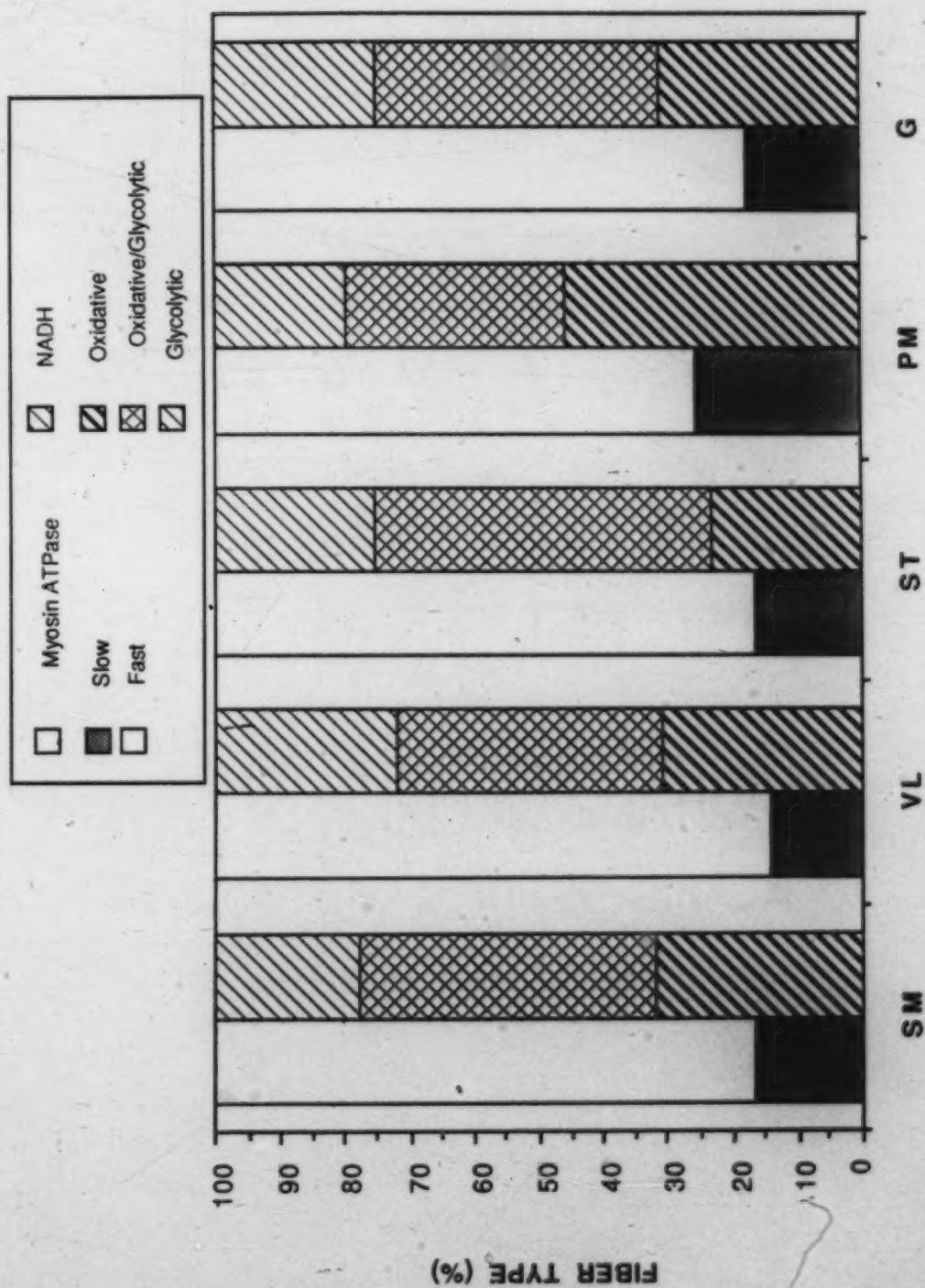


Figure V.19 Average fiber type for all five muscles using both the myosin ATPase and NADH dehydrogenase stains

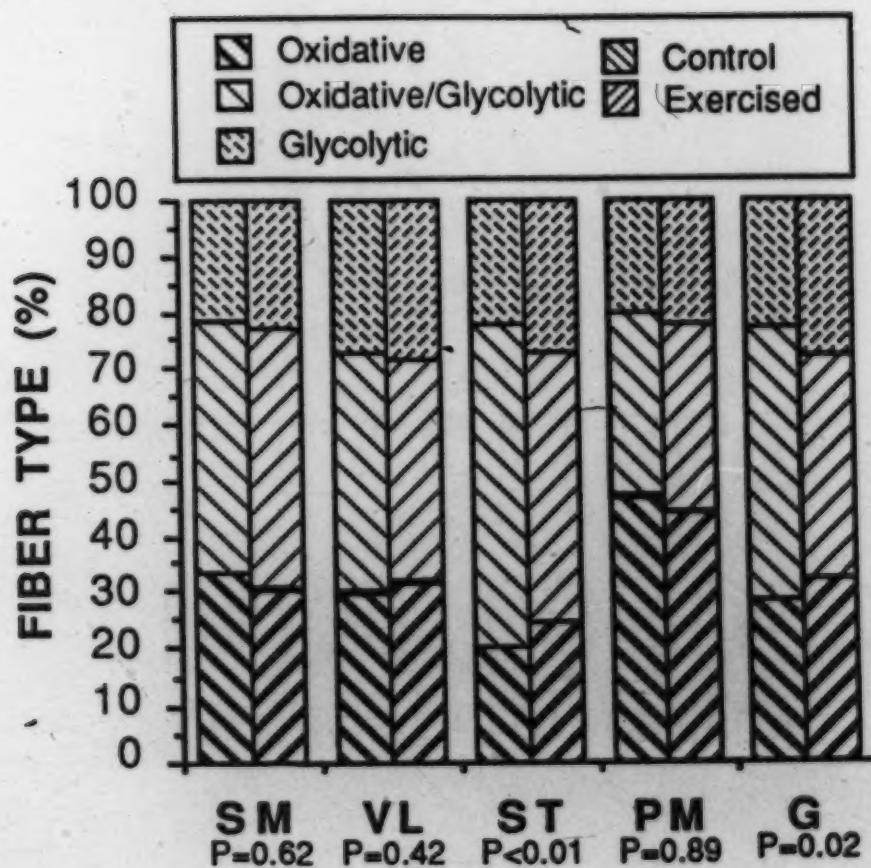
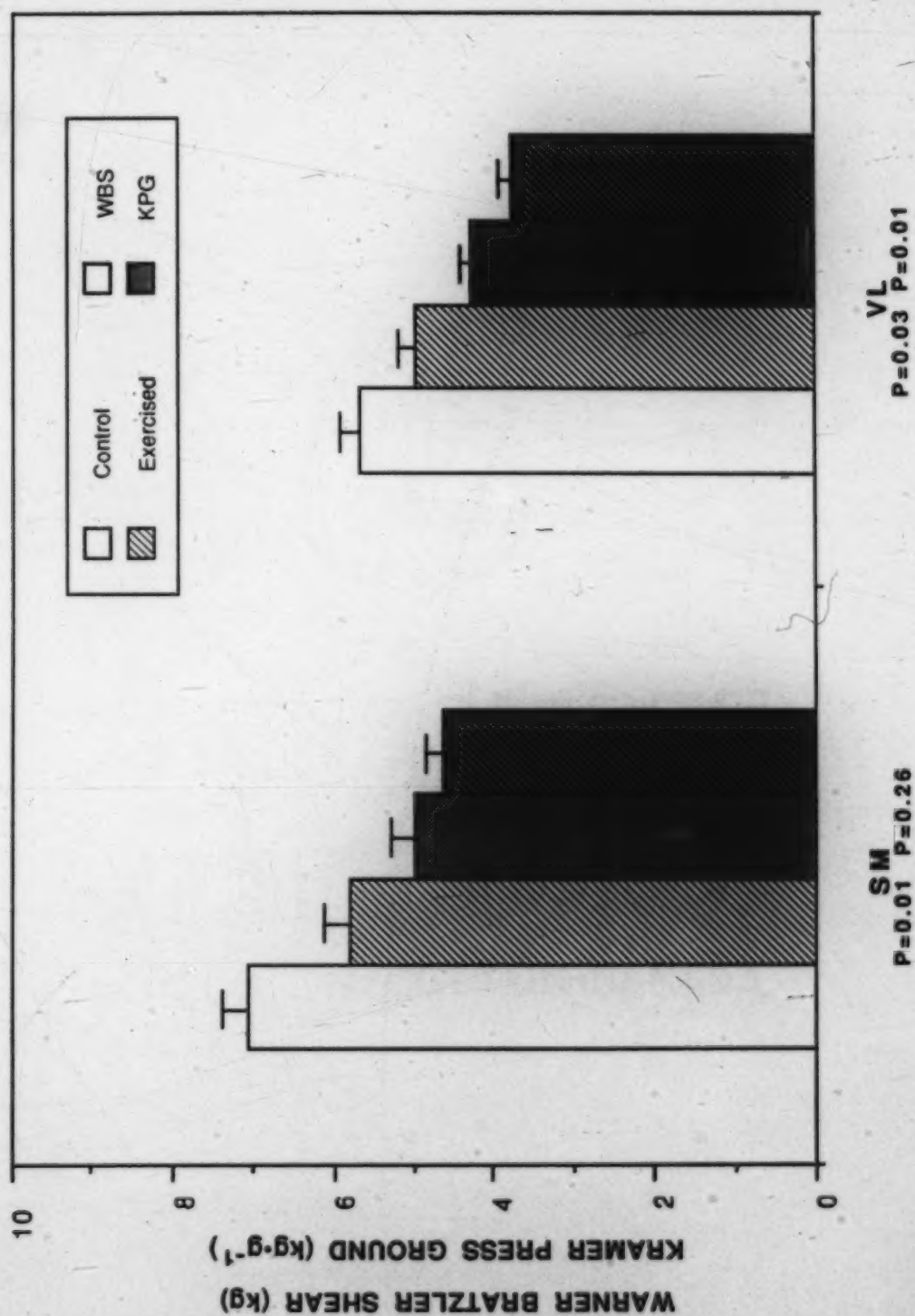


Figure V.20 Mechanical tenderness of the *semimembranosus* and *vastus lateralis* muscles from control and endurance-exercised sheep split by treatment



The fresh weights of the SM and VL were slightly, but non-significantly greater in the exercised sheep than in the control sheep (Table V.17). SM muscles from exercised sheep also tended to have lower thaw drip losses (4.6 vs. 6.0%; $P=0.06$), contributing to slightly heavier thawed weights (604 vs. 562 g; $P=0.09$). The fresh weights of the SM and VL increased significantly over the three slaughter groups ($P=0.02$, $P=0.04$). For both the SM and VL, as thawed weight increased over the three slaughter groups ($P=0.01$, $P=0.04$, respectively) the percentage of thaw drip tended to decrease. The heavier SM also tended to have higher overall drip losses than the VL.

There were several differences between control and exercised sheep in the cooking characteristics of the SM and VL muscles. The difference in trimmed raw weight between exercised and control sheep was due in part to the initial difference in thawed weight between the two treatments and in part to differences in sample weight taken for the ultimate pH measurements (Table V.18). The percentage of cooking drip was significantly lower in the SM from exercised sheep than in the SM from control sheep (3.1 vs. 3.8%; $P=0.04$). This contributed to the significantly higher cooked weight in the exercised SM. Similarly, trimmed raw weight and cooked weight increased significantly in both the SM and VL over the three slaughter groups. The percentage of cooking drip was significantly different among slaughter groups in the SM but there was no distinguishable trend (3.4, 4.0, 3.0 %; $P=0.05$). The percentage of volatile drip in the SM decreased significantly with increasing muscle weight over the three slaughter groups. There were no significant differences in the percentages of cooking drip or volatile drip among the three slaughter groups for the VL. Overall, the VL tended to have higher cooking drip losses than and similar volatile drip losses to the SM.

Chemical characteristics

Exercised and control sheep did not differ significantly in the chemical composition of the VL and G (Table V.19). However, the chemical composition of the SM tended to differ between exercised and control sheep; the SM from exercised sheep tended to have a slightly higher fraction of water (76.7 vs. 76.0 %; $P=0.08$), a significantly higher proportion of

Table V. 17 Thawing characteristics of the semimembranosus and vastus lateralis muscles from control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment			Probability
	Control	Exercised		
Number of animals	11	11	-	
Semimembranosus				
Fresh weight, g.	596±16.9	631±16.9	0.16	
Thawed weight, g.	563±16.4	605±16.4	0.09	
Thawing drip, %	6.0±0.50	4.6±0.50	0.06	
Vastus lateralis				
Fresh weight, g.	297±10.9	303±10.9	0.73	
Thawed weight, g.	293±11.0	299±11.0	0.71	
Thawing drip, %	1.7±0.25	1.5±0.25	0.49	
Slaughter Group				
	6 Weeks	12 Weeks	18 Weeks	Probability
Number of animals	6	8	8	-
Semimembranosus				
Fresh weight, g.	556±22.9 ^a	618±19.8 ^{a,b}	651±19.8 ^b	0.02
Thawed weight, g.	524±22.2 ^a	589±19.2 ^b	623±19.2 ^b	0.01
Thawing drip, %	6.3±0.67	5.3±0.58	4.6±0.58	0.18
Vastus lateralis				
Fresh weight, g.	265±14.7 ^a	309±12.8 ^b	316±12.8 ^b	0.04
Thawed weight, g.	261±14.8 ^a	306±12.8 ^b	312±12.8 ^b	0.04
Thawing drip, %	2.0±0.34	1.3±0.29	1.6±0.29	0.30

a, b Means not followed by the same letter are significantly different at the 5% level of probability. There were no significant treatment by slaughter group interactions for any of these traits.

Table V. 18 Cooking characteristics of the *semimembranosus* and *vastus lateralis* muscles from control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		
	Control	Exercised	Probability
	11	11	-
Number of animals			
<i>Semimembranosus</i>			
Trimmed raw weight, g	529±16.0	573±16.0	0.07
Cooked weight, g	395±12.1	439±12.1	0.02
Cooking drip, %	3.8±0.21	3.1±0.21	0.04
Volatile drip, %	21.6±0.68	20.4±0.68	0.24
<i>Vastus lateralis</i>			
Trimmed raw weight, g	270±10.8	277±10.8	0.65
Cooked weight, g	198±6.5	204±6.5	0.51
Cooking drip, %	6.2±0.38	5.7±0.38	0.37
Volatile drip, %	20.3±0.64	20.2±0.64	0.97
Slaughter Group			
	6 Weeks	12 Weeks	18 Weeks
Probability			
Number of animals			
<i>Semimembranosus</i>			
Trimmed raw weight, g	494±21.7 ^a	555±18.8 ^{a,b}	590±18.8 ^b
Cooked weight, g	369±16.3 ^a	409±14.1 ^a	461±14.1 ^b
Cooking drip, %	3.4±0.29 ^{a,b}	4.0±0.25 ^a	3.0±0.25 ^a
Volatile drip, %	21.9±0.91 ^a	22.3±0.79 ^a	19.0±0.79 ^b
<i>Vastus lateralis</i>			
Trimmed raw weight, g	242±14.6	281±12.7	288±12.7
Cooked weight, g	182±8.8 ^a	203±7.6 ^{a,b}	213±7.6 ^b
Cooking drip, %	5.6±0.51	6.1±0.44	6.0±0.44
Volatile drip, %	19.3±0.86	21.4±0.75	19.9±0.75

^{a,b} Means not followed by the same letter are significantly different at the 5% level of probability. There were no significant treatment by slaughter group interactions for any of these traits.

Table V.19 Proximate analysis of the semimembranosus, vastus lateralis and gastrocnemius muscles from control and endurance-exercised sheep split by treatment.

	Treatment		Probability
	Control	Exercised	
Number of animals	11	11	-
<i>Semimembranosus</i>			
Water, %	76.0±0.2	76.7±0.2	0.08
Protein, % [†]	78.3±1.1	81.5±1.1	0.05
Fat, % [†]	13.1±0.9	10.5±0.9	0.06
Ash, % [†]	3.4±0.09	3.2±0.09	0.15
Energy, KJ.g ⁻¹ [†]	22.9±0.16	22.5±0.16	0.08
<i>Vastus lateralis</i>			
Water, %	76.3±0.2	76.3±0.2	0.92
Protein, % [†]	75.8±1.1	74.7±1.1	0.48
Fat, % [†]	14.8±0.6	15.3±0.6	0.51
Ash, % [†]	3.9±0.11	4.1±0.11	0.54
Energy, KJ.g ⁻¹ [†]	23.4±0.12	23.4±0.12	0.66
<i>Gastrocnemius</i>			
Water, %	76.3±0.2	76.3±0.2	0.79
Protein, % [†]	80.7±1.1	78.1±1.1	0.11
Fat, % [†]	11.1±0.4	11.2±0.4	0.89
Ash, % [†]	3.6±0.09	3.6±0.09	0.88
Energy, KJ.g ⁻¹ [†]	22.7±0.08	22.8±0.08	0.34

[†] Dry matter basis.

protein (81.5 vs. 78.3 %; $P=0.05$), and a slightly lower fat content (10.5 vs. 13.1; $P=0.06$). There were no significant differences among slaughter groups in the composition of the SM, VL or G but there was a significant treatment by slaughter group interaction for fat content (Figure V.21). VL and G muscles from exercised sheep had a significantly smaller percentage of fat in the third slaughter group. The energy content of the muscles closely paralleled their fat content. The SM from exercised sheep had slightly less energy than its control counterpart (22.5 vs. 22.9 KJ.g⁻¹; $P=0.08$). Energy content was similar between the two treatments in the VL and G. In addition, energy content closely paralleled the treatment by slaughter group interaction changes in fat content (Figure V.22 compared to Figure V.21).

Exercised sheep did not differ from control sheep in the content of total protein, myofibrillar protein or total collagen in the SM (Table V.20). However, the SM from exercised sheep did tend to have less soluble collagen (37.0 vs. 42.2 mg; $P=0.07$). Myofibrillar protein:total collagen ratios and myofibrillar fragmentation indices were similar between the SM from control and exercised sheep. The amount of soluble collagen decreased significantly over the three slaughter groups (47.3, 37.8, 35.6 mg; $P=0.01$) whereas the MFI tended to increase over the three slaughter groups (11.1, 16.9, 15.9; $P=0.07$).

Exercised sheep had significantly more myofibrillar protein (611 vs. 570 mg; $P=0.01$) and significantly less total collagen (5.93 vs. 6.74 mg.g⁻¹) in the VL than control sheep (Table V.21). This resulted in a significantly higher myofibrillar protein to total collagen ratio in the VL from exercised sheep than in the VL from control sheep (18.8 vs. 14.4; $P<0.01$). The amount of total collagen tended to increase ($P=0.08$), and the amount of soluble collagen decreased ($P=0.01$) over the three slaughter groups. The myofibrillar protein to total collagen ratio was significantly higher in the 12 week slaughter group than in either the 6 or 18 week slaughter groups ($P=0.02$).

Total protein and myofibrillar protein content did not vary significantly between G muscles from exercised and control sheep (Table V.22). However, the G from exercised sheep tended to have lower residual glycogen content (48 hours post-mortem) than the G muscles from control sheep. A slightly, but non-significantly, higher ultimate pH was

Table V.20 Protein, collagen and myofibrillar fragmentation index (MFI) values for the semimembranosus muscle from control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		
	Control	Exercised	Probability
Number of animals	11	11	-
Total protein, mg.g ⁻¹ †	176±6.4	185±6.4	0.32
Myofibrillar protein, mg ††	563±16.1	578±16.1	0.53
Total collagen, mg.g ⁻¹ †	6.15±0.35	6.15±0.35	0.92
Soluble collagen, mg ‡	42.2±1.9	37.0±1.9	0.07
Myofibrillar protein:total collagen ratio	16.4±1.3	18.0±1.3	0.40
MFI [*]	15.2±1.33	14.7±1.33	0.79
Slaughter Group			
	6 Weeks	12 Weeks	18 Weeks
Number of animals	6	8	8
Total protein, mg.g ⁻¹ †	169±8.7	189±7.5	181±7.5
Myofibrillar protein, mg ††	591±21.7	561±18.8	564±18.8
Total collagen, mg.g ⁻¹ †	6.07±0.47	5.95±0.41	6.49±0.41
Soluble collagen, mg ‡	47.3±2.6 ^a	37.8±2.2 ^b	35.6±2.2 ^b
Myofibrillar protein:total collagen ratio	17.0±1.7	18.4±1.5	16.2±1.5
MFI [*]	11.1±1.80	16.9±1.56	15.9±1.56

† Frozen weight basis.

†† Per gram of total protein.

‡ Per gram of total collagen.

* Absorbance per 0.5 mg myofibrillar protein x 200.

^{a, b} Means not followed by the same letter are significantly different at the 5% level of probability.

There were no significant treatment by slaughter group interactions for any of these traits.

Table V.21 Protein, collagen and myofibrillar fragmentation index (MFI) values for the *vastus lateralis* muscle from control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		
	Control	Exercised	Probability
Number of animals	11	11	-
Total protein, mg.g ⁻¹ †	170±5.9	178±5.9	0.35
Myofibrillar protein, mg ††	570±10.4	611±10.4	0.01
Total collagen, mg.g ⁻¹ ‡	6.74±0.26	5.93±0.26	0.05
Soluble collagen, mg.g ⁻¹ §	43.7±1.9	42.8±1.9	0.75
Myofibrillar protein:total collagen ratio	14.4±1.0	18.8±1.0	<0.01
MFI ¶	14.2±1.29	14.1±1.29	0.94
Slaughter Group			
	6 Weeks	12 Weeks	18 Weeks
Number of animals	6	8	8
Total protein, mg.g ⁻¹ †	162±7.9	185±6.9	173±6.9
Myofibrillar protein, mg ††	582±14.1	606±12.2	582±12.2
Total collagen, mg.g ⁻¹ ‡	6.14±0.36	5.89±0.31	6.93±0.31
Soluble collagen, mg.g ⁻¹ §	50.5±2.6 ^a	41.6±2.2 ^b	39.5±2.2 ^b
Myofibrillar protein:total collagen ratio	15.4±1.4 ^a	19.7±1.2 ^b	14.5±1.2 ^a
MFI ¶	13.0±1.74	13.7±1.51	15.4±1.51
			0.11
			0.33
			0.08
			0.01
			0.02
			0.55

† Frozen weight basis.

†† per gram of total protein.

‡ per gram of total collagen.

§ Absorbance per 0.5 mg myofibrillar protein X 200.

a, b Means not followed by the same letter are significantly different at the 5% level of probability.

There were no significant treatment by slaughter group interactions for any of these traits.

Table V.22 Chemical characteristics of the *gastrocnemius* muscle from control and endurance-exercised sheep split by treatment and by slaughter group.

	Treatment		Probability
	Control	Exercised	
Number of animals	11	11	-
Total protein, mg.g ⁻¹ †	181±5.0	177±5.0	0.58
Myofibrillar protein, mg ††	573±8.1	583±8.1	0.36
Glycogen, mg.g ⁻¹ ‡	31.4±1.77	26.7±1.77	0.07
Ultimate pH	5.76±0.047	5.84±0.047	0.27
Slaughter Group			
	6 Weeks	12 Weeks	18 Weeks
Number of animals	6	8	8
Total protein, mg.g ⁻¹ †	177±6.8	179±5.9	181±5.9
Myofibrillar protein, mg ††	578±11.0	590±9.5	566±9.5
Glycogen, mg.g ⁻¹ ‡	26.5±2.40	33.4±2.07	26.6±2.07 ^b
Ultimate pH	5.52±0.063 ^a	5.90±0.055 ^b	5.92±0.055 ^b
			0.91
			0.24
			0.06
			<0.01

† Frozen weight basis.

†† Per gram of total protein.

‡ Forty-eight hours post-mortem, per gram of fresh weight.

• Twenty-four hours post-mortem, measured with a Corning pH meter.

a, b Means not followed by the same letter are significantly different at the 5% level of probability.

There were no significant treatment by slaughter group interactions for any of these traits.

Figure V.21 Treatment by slaughter group interactions for percentage chemical fat in the *semimembranosus*, *vastus lateralis* and *gastrocnemius* muscles

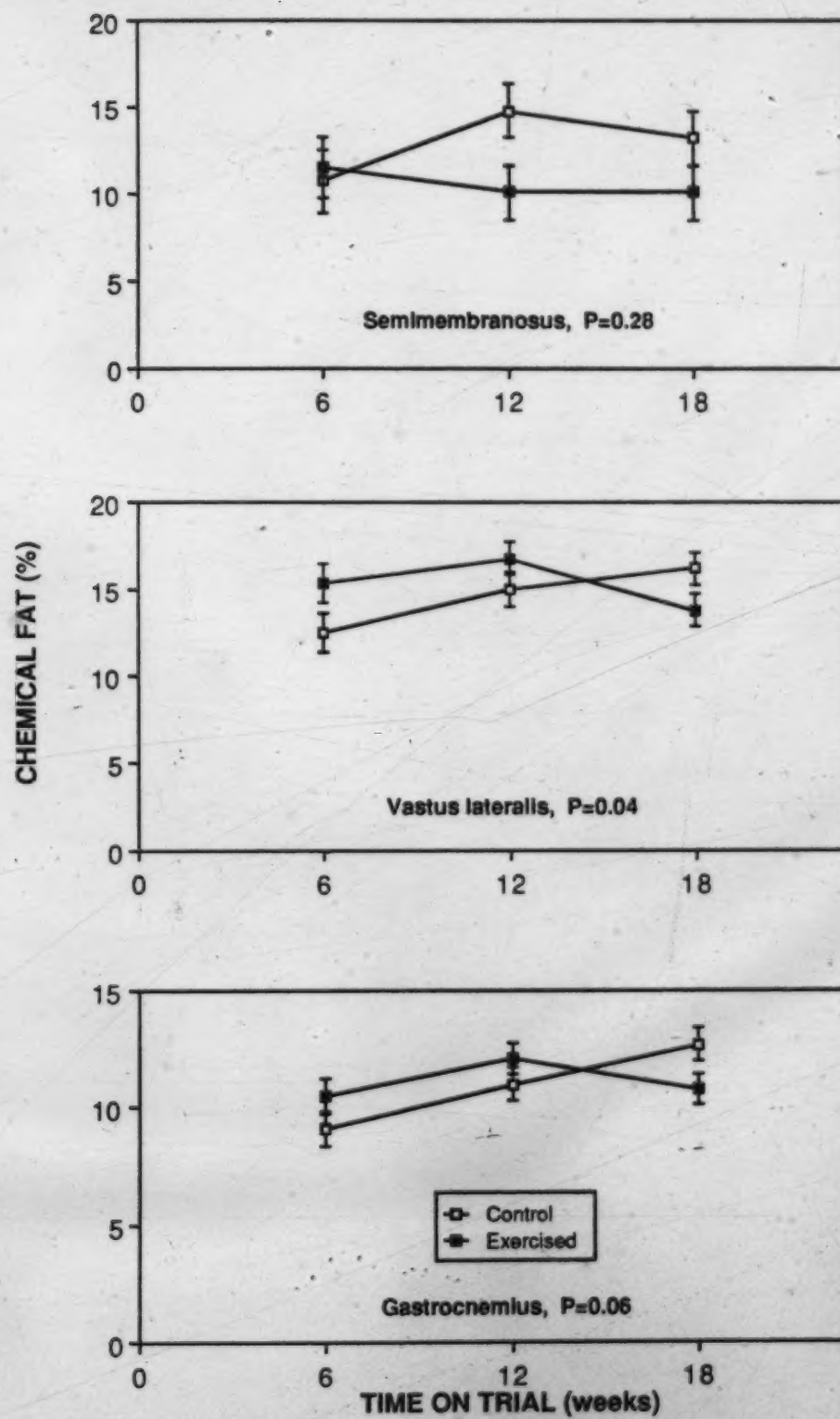
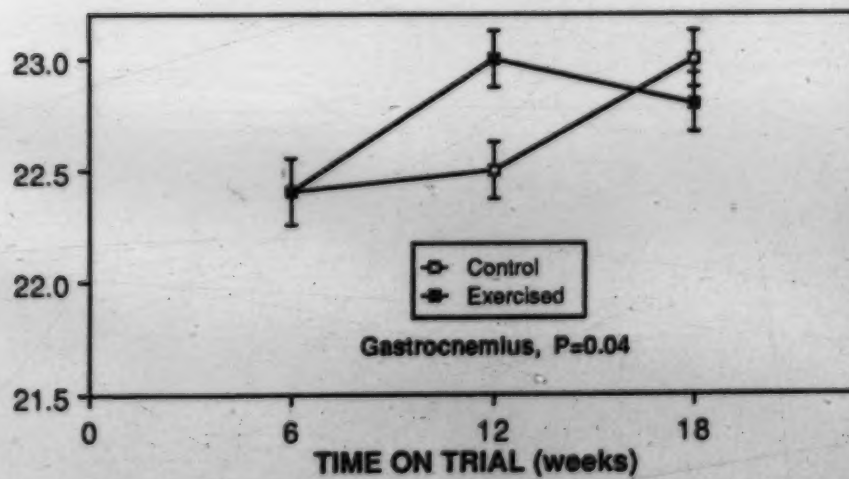
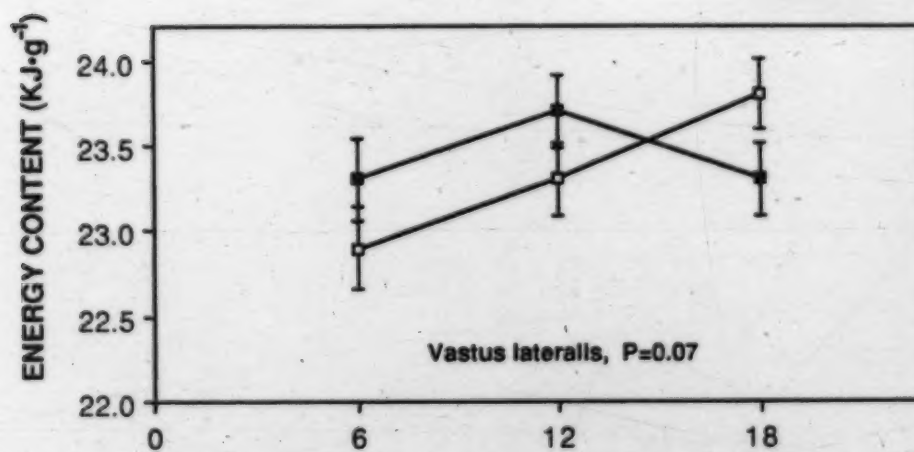
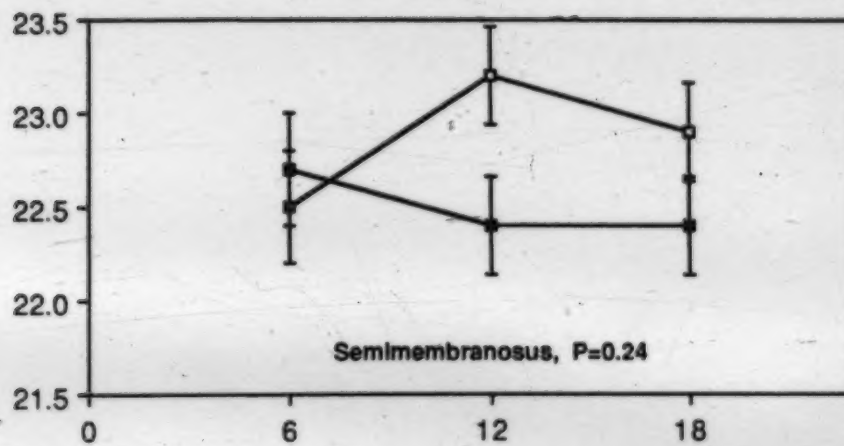


Figure V.22 Treatment by slaughter group interactions for energy content in the *semimembranosus*, *vastus lateralis* and *gastrocnemius* muscles



obtained 24 hours post-mortem in the G from exercised sheep than in the G from controls (5.84 vs. 5.76). The 12 week slaughter group had higher levels of residual glycogen than either the 6 or 18 week slaughter groups ($P=0.06$). The 6 week slaughter group had a significantly lower 24 hour ultimate pH than either the 12 or 18 week slaughter groups ($P<0.01$).

D. DISCUSSION

Oxygen consumption

Differences in the ability of individual sheep to perform the treadmill exercise were reflected in the significant animal effect on VO_2 max and net oxygen consumption. To compensate, training loads for the sheep in this experiment were set at 75% of their VO_2 max, slightly above the lactate threshold for trained athletes. The lactate threshold is defined as the point at which increasing intensity of the exercise results in a rapid rise in blood lactic acid levels. During low intensity exercise, blood lactic acid levels remain unchanged from resting values (Davis, 1985). In untrained humans the lactate threshold is reached at approximately 50-60% VO_2 max (Davis *et al.*, 1976, 1979), and at 70-80% VO_2 max in trained athletes (Farrell *et al.*, 1979).

There were no significant changes in VO_2 max or net oxygen consumption in response to the training program in this experiment. Other researchers have reported no change (Fox *et al.*, 1975a) or a slight decrease (Ekblöm *et al.*, 1968; Fox *et al.*, 1975b) in VO_2 max as a result of submaximal training. An increase in mechanical efficiency in performing the exercise may well have contributed to the decrease in VO_2 max. However, the slight downward trend in VO_2 max over time is very similar to the decrease in VO_2 max seen in children (Mirwald and Bailey, 1986) and may be a normal growth pattern.

Resting oxygen consumption (pre-exercise) decreased with time on trial. The decrease in resting oxygen consumption is probably primarily due to increased familiarity with the treadmill regimen. The initial high stress levels could have resulted in false resting oxygen

consumption values. In addition, Fox and Mathews (1981) reported anticipatory increases in oxygen consumption prior to exercise; this anticipatory rise in oxygen consumption may have decreased as training progressed in this study.

Live animal performance

Decreased feed intake in treadmill-exercised farm animals has been reported by Mitchell and Hamilton (1933), Bull and Rusk (1942) and Spaeth *et al.* (1967). In contrast, exercised sheep in this experiment had significantly higher feed intakes than control sheep. Exercised sheep also tended to have slightly higher average daily gains which resulted in higher overall weight gains. Most other researchers noted lower average daily gains (Mitchell and Hamilton, 1933; Spaeth *et al.*, 1967; Morrison *et al.*, 1968), or no difference in average daily gains (Peo *et al.*, 1970; Mandigo *et al.*, 1971) in exercised animals. This apparent contradiction may result from differences in intensity of the exercise (Stevensen *et al.*, 1966; Crews *et al.*, 1969). Stevenson *et al.* (1966) found that low intensity exercise resulted in a significant decrease in feed intake in rats while higher levels of exercise resulted in a proportionate increase in food intake. Crews *et al.* (1969) suggested that the mechanism responsible for the decrease in feed intake was increased secretion of adrenalin mediated through glucostatic, thermostatic or lipostatic mechanisms (Stevensen *et al.*, 1967). Stevenson *et al.* (1967) also found a decrease in spontaneous activity in rats forced to perform an exercise, even to the point at which they were less active than non-exercised rats. The increase in feed intake and reduction in total activity could result in higher average daily gains in exercised animals.

Body composition changes

The slightly higher proportion of liver in the exercised sheep was probably due to the increased feed intake in the exercised sheep (Murray and Slezacek, 1980; Price and White, 1985). Hale *et al.* (1986) claimed a significant decrease in the proportion of liver as a result of treadmill exercise in barrows; they also reported a significantly lower feed intake in the

barrows. The greater proportion of fleece in exercised sheep may be due to differences in peripheral blood flow. Peripheral blood flow is stimulated during exercise as a thermoregulatory mechanism (Wyndham, 1973; Rowell, 1974), and may stimulate fleece growth.

Most internal organs grow more slowly than the body as a whole (Kirton *et al.*, 1972); they will decrease as a proportion of empty body weight as empty body weight increases. The lungs, liver and kidneys all decreased as a proportion of empty body weight over the three slaughter groups.

Normal growth changes in the proportion of muscle, fat and bone occurred as carcass weight increased throughout the experiment (Berg and Butterfield, 1976). Comparable full carcass dissections in exercise studies are limited. However, using non-invasive techniques, most endurance exercise studies in humans have shown decreased body fat and increased lean tissue (Wilmore *et al.*, 1970; Boileau *et al.*, 1971; Després *et al.*, 1985). Full carcass dissections in this experiment and of treadmill-exercised pigs (Murray *et al.*, 1974a) have shown no differences in the proportion of muscle, fat or bone.

The proportion of connective tissue was significantly lower in exercised sheep, primarily due to a decreased growth rate of the *ligamentum nuchae*, and to a lesser extent, to decreased growth of other tendons. It may be that exercise has a larger effect on elastin metabolism than on collagen metabolism, since the *ligamentum nuchae* contains large amounts of elastin. However, the mechanism responsible for the observed changes in connective tissue is not known, and no similar findings have been reported. Increases in the amount of dissectible connective tissue have been reported under conditions of compensatory hypertrophy (Tipton *et al.*, 1975; Kiiskinen, 1977; Souminen *et al.*, 1980).

Total muscle weight increased with very little change in muscle weight distribution as live weight increased over the three slaughter groups, as found by Richmond and Berg (1971). Total muscle weight did not increase as a result of endurance exercise. Thus, any muscular "hypertrophy" would have been at the expense of muscle weight loss in other muscles. Exercised sheep had larger proportions of muscle in the proximal pelvic limb and abdominal

wall, at the expense of muscles around the spinal column.

The increase in proportion of muscle in the proximal pelvic limb appeared to be a direct response to exercise. The *biceps femoris* and *semimembranosus* muscles are the two largest muscles in the proximal pelvic limb (Group 1); together they account for approximately 10% of total muscle mass and were significantly larger proportions of total muscle weight in the exercised sheep. The *gluteus medius* is the third largest muscle in the proximal pelvic group, and it also tended to be a larger proportion of total muscle mass in the exercised sheep. All three muscles are strong extensors of the hip joint; the *biceps femoris* functions additionally in extension of the stifle and hock joints and the *gluteus medius* functions additionally in abduction of the limb and rotation of the femur (May, 1964). Thus, these muscles were actively involved in the treadmill exercise.

The increase in proportion of abdominal wall muscle may have been due to the passive demands of increased visceral weight, due to a later stage of maturity or due to the active demands of expiration during exercise. Visceral weight may have increased as a result of the higher feed consumption in exercised sheep (Butterfield *et al.*, 1966; Butterfield and Johnson, 1971). However, the increase in proportion of the abdominal wall may also be attributed to a more mature distribution, since the proportion of abdominal wall increases as total muscle increases. The *obliquus externus abdominus* and the *transversus abdominus* were the larger abdominal wall muscles. Both are involved in supporting the abdominal viscera; however, they have secondary functions in assisting the forced expiration of air (May, 1964; Fox and Mathews, 1981).

In contrast the *longissimus*, the *psoas minor*, the *spinalis dorsi* and the *longissimus cervicis* all tended to be smaller proportions of total muscle in the exercised sheep. These muscles function as extensors and flexors of the back and neck and would have been used to a much lesser extent than the proximal pelvic limb muscles in the treadmill exercise (May, 1964). The spinal muscles, as a group, normally grow at the same rate as total muscle (Butterfield and Berg, 1966b). Perhaps the muscles actively involved in the exercise had a preferential uptake of nutrients, depleting the nutrient supply to the spinal muscles and

slowing their growth.

Bone distribution varied between the control and endurance-exercised sheep, even though total bone weight remained constant. In contrast, as total bone weight increased over the three slaughter groups, bone distribution did not vary. The decreased proportion of cervical vertebrae in endurance-exercised sheep remarkably parallels changes one would expect had the exercised sheep been of a smaller mature strain. Butterfield and Thompson (1983) reported that the cervical vertebrae matured at the same rate as total bone in the large strain Merino rams but were late maturing in the small strain Merino rams. Although the tarsus also differed in proportion between the two strains of rams, these differences were expressed at an early stage of maturity. At a late stage of maturity (as in this experiment), only the difference in the proportion of cervical vertebrae would be apparent.

Endurance exercise is believed to decrease total fat in adult humans but dissectible fat did not decrease in this study. Researchers working with humans have reported decreases in fat as a result of endurance exercise, based on hydrostatic weighing and skinfold measurements (Allen *et al.*, 1956; Wilmore *et al.*, 1970; Pollock *et al.*, 1975). Crews *et al.* (1969) reported a decrease in fat in whole body chemical analysis of endurance-exercised rats. Both hydrostatic weighing and chemical analysis include the intramuscular fat depot and fat deposited around the internal organs, which is not included in the dissectible carcass fat reported in this study. However, chemically determined fat was consistently lower in the SM from exercised sheep and lower in the VL and G from exercised animals from the latter two slaughter groups. This would account for some of the differences between chemically determined total fat and total dissectible fat.

Although the exercise used in this study did not alter either total fat weight or fat distribution, the proportion of subcutaneous fat did increase very rapidly between the 6 and 12 week slaughter groups. This proportionate increase did not occur at the expense of other depots and may indicate exercise-induced acceleration of subcutaneous fat growth. Exercise-related acceleration of growth in actively growing tissues has been proposed by Malina (1969).

Researchers using farm animals have indicated an increase in subcutaneous fat as a result of exercise (Spaeth *et al.*, 1967 and Mandigo *et al.*, 1971). In contrast, a recent study by Deprés *et al.* (1985) reported a decrease in the subcutaneous fat depot (based on skinfold measurements) as a result of aerobic training in humans. Neither Spaeth *et al.* (1967) nor Mandigo *et al.* (1971) carried out total dissections to obtain total fat weight. The increased subcutaneous fat depot may have resulted from accelerated deposition of subcutaneous fat similar to the results of this experiment.

Changes in muscle fiber volume and their relation to cellular hypertrophy

A wide range of muscle fiber diameters occurred within and among the five muscles studied. Similar to the results of Hammond (1932), the muscles with a dominant postural function (PM) had smaller mean fiber diameters than muscles with a major propulsive function (SM and VL). However, there was no evidence of distinct fiber populations within a muscle (based on the frequency distribution of the diameters), despite the fact that red and white fibers can differ in diameter at certain stages of development (Moody *et al.*, 1980; Solomon *et al.*, 1981) and that exercise can selectively affect fiber population diameters (Fox and Mathews, 1981). Unfortunately, the wide range in fiber diameters within a muscle (Tuma *et al.*, 1962; Solomon *et al.*, 1981) often masks a bimodal distribution (Swatland, 1984).

Sarcomere lengths varied among muscles in the carcass depending on the anatomical position and degree of stretch of the muscles in the Achilles-hung carcass. Muscles along the vertebral column (PM) and in the posterior lower hindlimb were stretched resulting in longer sarcomere lengths (McCrae *et al.*, 1971; Hostetler *et al.*, 1972; Swatland, 1984). In contrast, muscles in the posterior portion of the upper hindlimb were freed from skeletal restraint (SM) resulting in shorter sarcomere lengths. Anatomically deeper muscles in the upper hindlimb (ST) had longer sarcomeres than the more superficial muscles (VL) due to their larger role in carcass support (Hostetler *et al.*, 1972).

Changes in sarcomere lengths over the three slaughter groups did not follow the pattern I had expected. Since sarcomeres generally only elongate in very young animals (Swatland, 1984), further changes should have reflected anatomical position during hanging; as carcass weight increased, sarcomere lengths in stretched muscles should have increased. However, there was no distinct pattern since sarcomere lengths in the PM (a stretched muscle) shortened with increasing carcass weight, and sarcomere lengths in the SM (an unstretched muscle) lengthened with increasing carcass weight. This unpredictable response of sarcomere length to increasing carcass weight probably reflects changes in the cooperative action of adjacent muscles in supporting the carcass, as the muscles change in size and relative location during growth (Honikel *et al.*, 1986). In addition, the age-related increase in cross-linking in the connective tissue (Goll *et al.*, 1962) may have made the myofiber more resilient to changes in sarcomere length.

Based on the unadjusted fiber diameters, muscular hypertrophy of the VL could be claimed as a result of endurance exercise. However, even though larger muscle fiber diameters have been used previously to indicate muscular hypertrophy (Gonyea and Ericson, 1976; McDougall *et al.*, 1979; Schantz *et al.*, 1981), fiber diameters must be interpreted with caution, since sarcomere length can affect fiber diameter (Swatland, 1984). Surprisingly, sarcomere lengths were shorter in the endurance-exercised sheep over all five muscles, which may have led to misleadingly large fiber diameters. Adjustment of the diameters to a constant sarcomere length within a muscle has been proposed by Swatland (1984) to overcome this problem. In the live animal where sarcomeres remain isovolumetric during contraction (Morel *et al.*, 1976; Heffron and Hegarty, 1974; Millman and Irving, 1981) this adjustment is justifiable. However, in the post-mortem state, sarcomeres may not remain isovolumetric; fiber diameters may decrease as a result of water loss from the intracellular space (Hegarty, 1970; Currie and Wolfe, 1983; Honikel *et al.*, 1986). In addition, Maughan and Godt (1981) reported increased intracellular water loss at shorter sarcomere lengths. Thus, in post-mortem muscle, shorter sarcomere lengths result in two opposing changes: fiber diameters are increased due to the increase in overlap of myosin and actin filaments and fiber diameters are

decreased due to the loss of water from the intramuscular spaces. It is not known which, if either, of these effects would dominate changes in fiber diameter post-mortem.

In general, fiber diameters increase with increasing carcass weight (Cross *et al.*, 1972; Moody *et al.*, 1980; Solomon *et al.*, 1981). The only major exception in this experiment was the ST muscle which did not increase in myofiber diameter with increasing carcass weight. Since radial growth had ceased, the continued increase in ST weight must have been due to increases in muscle fiber length (Swatland, 1984). This assumption is supported indirectly by data which show a steady decrease in apparent fiber number in the ST muscle in cattle from 11 days after birth to 2 years of age (Bendall and Voyle, 1967). As the ST muscle increases in length, some of the intrafascicularly terminating fibers would disappear, resulting in a decrease in apparent fiber number.

Changes in post-mortem pH decline and their relationship to sarcomere length

Post-mortem pH decline and ultimate pH can vary significantly from muscle to muscle within a carcass (Hendricks *et al.*, 1970; Bouton *et al.*, 1984; this experiment). The rate of pH decline among muscles is dependent on the initial glycogen stores of the muscle, the rate of temperature decline within the muscle (related to position in the carcass) and the ability of the muscle to break glycogen down (Jeremiah, 1978). Ultimate pH is inversely related to the initial energy stores (Honikel, 1976).

In this experiment, muscles from exercised sheep had lower rates of post-mortem pH decline than muscles from control sheep. Since muscle pH values are closely related to the rate of post-mortem glycolysis (Fredeen *et al.*, 1974), lower rates of pH decline post-mortem may indicate a slower post-mortem glycolysis. Stress before slaughter can deplete stored glycogen, resulting in low rates of post-mortem glycolysis and a high ultimate pH (Briskey *et al.*, 1959; Chrystall *et al.*, 1982). The exercised sheep in this experiment rested for 48 hours prior to slaughter and did not differ from controls in ultimate pH. Hence, it is unlikely that the lower rates of post-mortem glycolysis resulted from differences in energy stores before slaughter. Exercise-trained animals and humans typically have higher glycogen levels than

untrained animals (Pattengale and Holloszy, 1967; Lindberg *et al.*, 1973).

There are three possible explanations for the decreased rate of post-mortem pH decline in muscles from endurance-exercised animals: (1) lower activities of glycolytic enzymes in post-mortem muscle could result in a slower production of lactate and a lower rate of pH decline; (2) the decreased rate of pH decline in the exercised sheep may have been due to less stress-susceptibility at slaughter; (3) a change in buffering capacity in muscles from endurance-exercised sheep may affect the rate of pH decline. Each of these possibilities will be considered. Endurance training is reported to reduce the rate of glycogen breakdown and the resulting production of lactate (Lindberg *et al.*, 1973; Eriksson *et al.*, 1973), perhaps through a decrease in phosphorylase, phosphofructokinase (PFK; Gollnick *et al.*, 1973; Baldwin *et al.*, 1976, 1977), or lactate dehydrogenase (LDH) activity (Ianuzzo and Chen, 1979). Exercised sheep were more familiar with handling and strange surroundings than the control sheep, thus a rapid rate of glycolysis immediately prior to slaughter would probably not occur. Endurance exercise may have increased the buffering capacity of active muscles (Hodgson, 1985) and therefore slowed post-mortem pH decline. Muscle proteins account for approximately half the buffering capacity in the post-mortem pH range, whereas orthophosphate, carnosine and anserine account for the other half of the buffering capacity (Bate-Smith, 1938). Carnosine levels have been shown to increase as a result of anaerobic training (Parkhouse *et al.*, 1983).

Shorter sarcomere lengths in muscles from exercised sheep were probably due to exposure to lower temperatures before the onset of rigor (due to a slower rate of glycolysis post-mortem and possibly higher energy stores before slaughter). The pH decline reflects the loss of energy substrates post-mortem, the onset of rigor mortis, and the subsequent sarcomere length (Sink *et al.*, 1965; Bendall, 1973; Hamm, 1977). Rigor mortis develops when there is either insufficient ATP to break the myosin/actin crossbridges or when there is a loss of membrane integrity in the sarcoplasmic reticulum resulting in an influx of Ca^{+2} . Loss of membrane integrity is both temperature and pH dependent (Jeremiah, 1978); exposure of the muscle to either too cold a temperature, or too low a pH at high temperatures before the

onset of rigor can result in disruption of Ca^{+2} sequestration and increase sarcomere shortening. The lower pH decline in the exercised muscles indicates a slower development of rigor; exercised muscles appear to have been exposed to lower temperatures than control muscles before the onset of rigor. Both a greater store of ATP and glycogen (Ulyanov and Touleuov, 1976) and higher carnosine levels (Bowen, 1965) in the muscle have been implicated in a longer delay phase before the onset of rigor resulting in shorter sarcomere lengths.

In addition, sarcomere lengths may have been influenced by differences in the connective tissue between exercised and control sheep. Honikel *et al.* (1986) indicated that the degree of contraction of a muscle in a carcass is dependent on the structure of the connective tissue network within the muscle; increased cross-linking in the connective tissue may decrease the degree of myofiber shortening (Goll *et al.*, 1962). Since there was some indication that the connective tissue component may have differed between exercised and control sheep, it is possible that sarcomere length may have been influenced.

Changes in fiber type distribution and their relation to post-mortem pH decline and myofiber volumes

Muscle fiber types have been classified in a number of ways (Brooke and Kaiser, 1970; Tunnell and Hart, 1977; Ashmore *et al.*, 1977). The following types will be used in this discussion: (1) slow oxidative (SO), high oxidative capacity and slow myosin ATPase activity; (2) fast oxidative glycolytic (FOG), both glycolytic and oxidative activity with fast myosin ATPase activity; (3) fast glycolytic (FG), high glycolytic activity and fast myosin ATPase activity.

The five muscles in this experiment had similar fiber type distributions as reported previously by Suzucki (1971a,b) and White *et al.* (1978). Muscles with a dominant postural function (PM) had more SO fibers than muscles with a major propulsive function (ST). However, White *et al.* (1978) indicated that the proportion of FOG fibers decreases with age, and the proportion of FG fibers increases with age. Since the proportion of FG and FOG fibers for the VL muscle are within the range reported to occur at different ages by White *et*

al. (1978), it was assumed that the differences between my data and those of Suzucki (1971a,b) were due to an age difference.

Endurance-exercise slightly increased the proportion of SO fibers in the SM, VL, ST and G in this experiment. Although some researchers have reported similar increases in SO fibers as a result of exercise (Jaweed *et al.*, 1977; Moody *et al.*, 1980; Green *et al.*, 1984; Pette, 1984), other researchers have found no change in fiber type but an increase in oxidative enzyme activity (Barnard *et al.*, 1970; Baldwin *et al.*, 1972; Gollnick *et al.*, 1973; Eriksson *et al.*, 1973; Henriksson and Reitman, 1976). The apparent contradiction is probably due to different exercise intensities or lengths of time on exercise. Pette (1984) proposed an orderly sequence for this transformation, starting with early changes in enzyme and isoenzyme activities resulting in a glycolytic to oxidative metabolic transformation. The exchange of fast with slow type myosin isoforms occurs last.

The increase in SO fibers as a result of endurance-exercise remarkably parallels similar age-dependent changes found by White *et al.* (1978). Indeed, Syrový and co-workers (1972) indicated that the effects of exercise were more pronounced in muscles of young animals. Similarly, Tomanek (1975) found that the age associated conversion of fast to slow fibers was accelerated during compensatory hypertrophy in kittens but that there was no similar change in fiber type in adult cats. Thus, the increase in SO fibers may be another example of acceleration of normal growth patterns induced by exercise (Malina, 1969).

Since fiber type composition of a muscle may ultimately dictate all its properties (Cassens and Cooper, 1971; Anderson and Parrish, 1972; Ashmore, 1974; Cassens, 1977; Swatland, 1984), an attempt was made to relate fiber type composition to post-mortem pH decline and sarcomere lengths. Muscles with a high proportion of FG fibers might be expected to have a rapid post-mortem pH decline since they have high glycolytic and LDH activities. However, the opposite occurred; muscles with a high proportion of FG fibers (G and VL) had slow pH declines, whereas muscles with a low proportion of FG fibers (PM) had rapid pH declines. This apparent contradiction may be explained by the fact that muscles with a high glycolytic activity also have a high buffering capacity (Parkhouse *et al.*, 1983;

Hodgson, 1985) which would reduce the rate of pH decline. Differences in pH decline in muscles from exercised and control sheep were poorly related to fiber type distribution. Of the four muscles which had increased proportions of SO fibers, only one muscle (ST) had a significantly lower rate of pH decline in the exercised sheep. The slower pH decline in the ST could be due to an augmentation of skeletal muscle myoglobin through exercise (Pattengale and Holloszy, 1967) which may increase the aerobic potential immediately post-mortem and the delay before anaerobic glycolysis begins.

Among the five muscles studied, fiber type distributions can not be used as a predictor of sarcomere lengths since the degree of stretch during hanging would influence this relationship. However, between individual muscles from control and exercised carcasses the degree of stretch would be the same, hence fiber type distributions may be related to sarcomere lengths. Slow-oxidative fibers have shorter sarcomere lengths (Cassens and Cooper, 1971; Jeremiah *et al.*, 1972), perhaps due to a greater susceptibility to cold-shortening (Jeremiah, 1978). In addition, Davey and Gilbert (1975) indicated that cold shortening in pre-rigor muscle was largely dependent on physiological maturity; mature carcasses (more SO fibers) tended to cold shorten more than immature carcasses at the same temperature. The decrease in sarcomere lengths in muscles from exercised sheep are consistent with the shift towards a higher proportion of SO fibers.

Changes in meat quality

There is a popular perception that exercise increases meat toughness in animals (Smith and Carpenter, 1970). However, in this study, as in Chapter IV, mechanically measured tenderness definitely increased in exercised sheep. Other researchers have generally reported either an increase in meat tenderness following exercise (Mitchell and Hamilton, 1933; Bull and Rusk, 1942; Spaeth *et al.*, 1967) or no measurable change (Mandigo *et al.*, 1971; Hawrysh *et al.*, 1974). The mistaken belief that exercise causes tougher meat may be partly due to age-related toughening in range livestock. In addition, the larger amount of collagen (and tougher meat) from the so-called "heavily" used muscles (i.e. in the distal thoracic limb)

than from "lightly" used muscles (i.e. those in the proximal pelvic limb; Hiner *et al.*, 1955; Kruggel and Field, 1974; Swatland, 1984) is an adaptation to locomotion (Davies, 1981) rather than to exercise.

The myofibrillar proteins and surrounding connective tissue have been identified as the two main factors contributing to meat tenderness (Cover *et al.*, 1962a,b; Bouton and Harris, 1972; Bouton *et al.*, 1975; Møller, 1980-81). The contributions of myofibrillar protein and connective tissue to meat tenderness are extremely complex and can be modified by post-mortem conditions (Jeremiah, 1978; Asghar and Pearson, 1980). Thus, the effects of: (a) sarcomere length; (b) fiber diameter; (c) ultimate pH and post-mortem pH decline; (d) post-mortem aging; (e) water holding capacity; (f) chemical composition; and (g) fiber type, will be discussed in relation to the two main components of tenderness and to the increased tenderness in the exercised muscles.

(a) Sarcomere length mainly affects the myofibrillar component and is inversely related to tenderness (Smith and Carpenter, 1970; Bouton and Harris, 1972; Hostetler *et al.*, 1972). The inverse relationship between sarcomere length and tenderness is due to the increase in the number of actin-myosin crossbridges formed at lesser sarcomere lengths, and the degree of crossbridge formation is intimately related to the rate of glycolysis post-mortem, post-mortem temperature and the development of rigor (Jeremiah, 1978). Thus, shorter sarcomeres in the muscles from exercised sheep might have contributed to a tougher meat. Dutson *et al.* (1976) showed an increase in connective tissue softness with increasing sarcomere length in some muscles. Swatland (1984) attributed this to the increased tension on the connective tissue at longer sarcomere lengths which would yield to a force more rapidly than if there was some elasticity or slack to be taken up first. With respect to the sheep in this study, shorter sarcomeres in muscles from exercised sheep would again be expected to contribute to a tougher meat. However, sarcomere shortening of less than 20% does not result in tougher meat (Marsh and Leet, 1966); sarcomeres in muscles from exercised sheep were never more than 10% shorter than their control counterparts.

(b) Muscle fibers with large diameters have been associated with decreased tenderness (Smith and Carpenter, 1970; Cross *et al.*, 1972). Thus, the slightly larger diameters in the muscles from exercised sheep might have contributed to decreased tenderness. However, fiber diameters tend to be a poor predictor of tenderness when the effect of animal age is removed (Tuma *et al.*, 1962).

(c) The influence of pH on tenderness can be variable; both low (Bouton *et al.*, 1982; Dutson, 1983) and high (Marsh *et al.*, 1981; Bouton *et al.*, 1984) pH early post-mortem, when temperatures are high, have been associated with more tender meat. In addition, high ultimate pH and more tender meat were found in animals exercised immediately before slaughter (Briskey *et al.*, 1959; Chrystall *et al.*, 1982). In this study, there were no differences in ultimate pH, but muscles from exercised sheep had a higher pH values than controls at similar post-mortem temperatures.

(d) Greater meat tenderness at high pH and temperature may result from myofibrillar protein degradation, perhaps because high pH and temperature post-mortem are ideal conditions for calcium activated protease activity (Yu and Lee, 1986; Zeece *et al.*, 1986). The MFI index (Davy and Gilbert, 1969) did not differ between SM and VL muscles from exercised and control sheep suggesting that the difference in tenderness between the exercised and control muscles was not due to differences in post-mortem degradation of myofibrils.

(e) Increased water holding capacity in the SM muscle from exercised sheep was unexpected since shorter sarcomeres generally result in lower water holding capacity (Bouton *et al.*, 1972b). However, increased water holding capacity is consistent with the increased meat tenderness in the SM from exercised sheep (McClain and Mullins, 1969; Bouton *et al.*, 1972a,b). Perhaps the increased water holding capacity in the SM from exercised sheep was due to a muscle shape factor which could decrease the amount of water loss due to a decreased surface to volume ratio; as the SM muscle increased in weight, the percentage of

thaw drip decreased. The lower cooking drip losses may also reflect a change in surface to volume as muscle weight increased.

(f) The significant increase in chemically determined water content in the SM muscle from exercised sheep may occur as a result of the slight increase in protein content in the muscle, since myofibrillar proteins are responsible for 90 - 95% of the water binding capacity of the muscle (Goll *et al.*, 1977). The increase in protein content may also reduce water drip losses by increasing the amount of bound water (Forrest *et al.*, 1975). However, exercise can change the hydration state of a muscle (McMorris and Elkins, 1954); the differences in water content could have been a direct effect of the exercise. The SM muscle from exercised sheep also had a lower percentage of chemically determined fat than their control counterparts which may have been reflected in the lower cooking drip losses in the SM from exercised sheep (Moody *et al.*, 1980; Solomon *et al.*, 1981).

(g) Red fibers (SO) have longer sarcomeres (Cassens and Cooper, 1971), smaller fiber diameters (Gauthier, 1970), less glycogen (Vander *et al.* 1980), greater susceptibility to cold shortening (Jeremiah, 1978; Cornforth *et al.*, 1980), and thicker Z-lines which are less susceptible to Z-line degradation post-mortem (Goll *et al.*, 1970; Gann and Merkel, 1978). Given these studies, the higher proportion of SO fibers in the muscles from exercised sheep would contribute towards a tougher meat.

None of the factors discussed contribute significantly towards a consistently more tender meat from exercised sheep. However, reduced levels of collagen and increased levels of myofibrillar protein per gram of muscle in the VL of exercised sheep may have increased meat tenderness.

Although the decreased amount of collagen has been attributed merely to an exercise-induced 'dilution' of connective tissue, resulting from an increase in myofibrillar protein (Mitchell and Hamilton, 1933; Helander, 1961), there is some evidence to suggest that collagen metabolism may be affected by exercise. Visible collagen increased under conditions

of compensatory hypertrophy (Van Linge, 1962; Ianuzzo and Chen, 1979) and more highly exercised muscles have less collagen cross-linkages than quiescent muscles (Krüggel and Field, 1974). As connective tissue ages, stabilizing cross-links form (Bailey, 1972) which is apparent from the decrease in solubility of collagen over the three slaughter periods in the SM and VL muscles. The initial form of cross-linkage in collagen are labile aldimine bonds which are easily ruptured by pH changes and heat (Bailey, 1972). It is possible that the conditions in the muscle during exercise (pH lowered and temperature elevated) create an environment where the initial aldimine bonds in collagen are not stable. Perhaps the increased turnover of collagen could result in a decreased collagen content in muscles from exercised sheep.

The SM, although also more tender in exercised sheep, did not show the same decrease in collagen and increase in myofibrillar protein as the VL. Several authors have reported the existence of a tenderness gradient within large muscles (Khan *et al.*, 1973; Buege and Stouffer, 1974), perhaps due to differences in muscle temperature during post-mortem pH decline (Hunt and Hedrick, 1977). Since tenderness measurements were made only on the anatomically deeper portion of the SM and the samples for myofibrillar protein and collagen determinations were collected from the superficial SM, it is likely that the chemical determinations would not reflect changes in tenderness from the deeper portion of the SM.

E. CONCLUSION

Live animal

It is important to adjust the intensity of an exercise to the ability of an individual; there were substantial differences in the intrinsic ability of the sheep to perform a standard VO_2 max test.

Exercised sheep in this experiment ate more feed, and had slightly higher average daily gains than control sheep. Thus, exercise did not adversely affect production.

Carcass

Exercised sheep had slightly higher proportions of liver and fleece than their control counterparts. The larger proportion of liver is probably due to the higher feed intake in the exercised sheep (Murray and Slezacek, 1975; Price and White, 1985). The increased proportion of fleece may be related to an increased peripheral blood flow in an attempt to dissipate heat.

Total weights of dissectible muscle, bone and fat were essentially similar for exercised and control sheep. However, exercised sheep had a smaller proportion of dissectible connective tissue, especially a lighter *ligamentum nuchae*. The mechanism by which this occurs is not known.

Exercised sheep had slightly higher proportions of muscle in the proximal pelvic limb and in the abdominal wall, and a corresponding decrease in the proportion of spinal muscles. The increase in proportion of muscle in the proximal pelvic limb was likely a direct result of exercise. These muscles were used extensively in the treadmill exercise. The increase in proportion of muscle in the abdominal wall may have resulted either as a direct response to the exercise or as a response to the increased feed intake. As well, it may indicate a later stage of maturity in the exercised sheep.

Exercised sheep had smaller proportions of cervical vertebrae than controls, but few other differences in bone weight distribution were apparent. This difference was similar to differences which result from different stages of maturity in small and large strain Merino rams at the same live weight.

There were some decreases in intramuscular fat in exercised sheep and also a rapid increase in subcutaneous fat weight between the 6 and 12 week slaughter groups.

These changes in muscle, fat and bone distribution may result from an acceleration of growth induced by exercise (Malina, 1969).

Cellular

Muscles from exercised-sheep tended to have slower pH declines post-mortem; this resulted in shorter sarcomeres. This may have reflected differences in stress-susceptibility between the exercised and control sheep or, as a result of the endurance training, exercised muscles may have had different glycolytic enzyme activities or different buffering capacities than control muscles.

Exercised sheep tended to have slightly larger average fiber diameters than control sheep, although when adjusted for sarcomere length, these differences were reduced. Thus, using unadjusted fiber diameters as an indication of hypertrophy may result in biased conclusions.

Chronic endurance exercise resulted in a shift in fiber type from fast to slow contraction speed and a decrease in the intermediate oxidative capacity fibers. These changes are not different from changes in fiber type distribution which normally occur as a result of increasing chronological age.

Meat quality

Increased tenderness in exercised muscles resulted from an increase in the proportion of myofibrillar protein to total collagen. Mitchell and Hamilton (1933) attributed the lower collagen content to a dilution effect, whereby the myofibrillar protein increased and the collagen content remained the same. However, some evidence exists for both an increase in myofibrillar protein (Helander, 1961) and a decrease in collagen (Bailey, 1972; Kruggel and Field, 1974) as a result of exercise. Further research is needed to clarify this point.

Chapter VI

THE EFFECTS OF EXERCISE: CONCLUSIONS

A. LIVE ANIMAL PERFORMANCE

Although the energy cost of exercise is relatively low (Morrison *et al.*, 1968; Murray *et al.*, 1974a), stress associated with exercise can depress appetite (Stevenson *et al.*, 1966; Crews *et al.*, 1969) and may be deleterious to animal production. The energy cost of exercise can be measured in terms of feed required to produce animals of the same live weight; load-carrying sheep required 9% more feed than control sheep to maintain live weight (Chapter II) and endurance-exercised sheep consumed only 6% more feed to maintain a similar growth rate as control sheep (Chapter V). More stress was associated with high intensity exercise than with endurance exercise, since load-carrying sheep (Chapter 3) and progressive-resistance-exercised sheep (Chapter 4) had depressed appetites. The lower feed consumption, lower rates of gain and lower live weights at slaughter associated with high intensity exercise may be more costly in terms of production than the extra feed required to maintain normal growth rates in endurance-exercised sheep.

B. BODY COMPOSITION

Offal components

The proportion of liver appeared to be consistently affected by exercise. However, the proportion of liver has been shown to reflect differences in feed consumption (Murray and Slezacek, 1980; Price and White, 1985); higher feed consumption (Chapters II and V) and lower feed consumption (Chapter III) were associated with a corresponding increase and decrease in the proportion of liver in exercised sheep.

Under conditions of static load-carrying (Chapters II and III) the front feet were a significantly larger proportion of the offal. The load-carrying exercise was thought to have accelerated bone growth to a point at which bone distribution appeared to be at a later stage

of maturity (Butterfield *et al.*, 1983c). Similar changes did not occur as a result of progressive-resistance exercise or endurance exercise (Chapters IV and V). These exercise regimes were thought to have less influence on bone growth since the role of bone in support was minimal, compared to the role of muscle in movement during dynamic exercise.

Total muscle weight and muscle weight distribution

The results of these experiments point to a two-fold response of muscle to exercise in growing animals: a localized response to the exercise, and an overall acceleration of growth toward a pattern typical of mature muscle distribution.

The localized response to exercise may be termed muscular hypertrophy since individual muscles increased in proportion. However, this hypertrophy was at the expense of growth in muscles less involved in the exercise; there was no overall increase in total muscle weight. Static load-carrying sheep (Chapter II) had a shift in muscle distribution from muscles in the neck and spinal area to muscles in the proximal thoracic limb (specifically muscles involved in posture maintenance); the load-carrying sheep tended to stand in a braced position as the weight of the load increased. The progressive-resistance-exercise caused a shift in muscle weight distribution to the extensor muscles of the elbow joint, presumably due to the strong contraction needed to "pull" the sheep up onto the platform jump. The endurance-exercised sheep had a shift in muscle weight distribution from spinal, neck and thoracic muscles to muscles of the proximal pelvic limb, which were heavily used in the treadmill exercise.

Exercise also resulted in an overall acceleration of growth toward a mature muscle distribution. The proportion of muscle weight in the abdominal muscles was larger in exercised sheep (Chapters II, III and V). These changes were not consistently associated with feed intake or exercise. However, the increase in proportion of abdominal muscles was very similar to the changes in muscle distribution associated with increased live weight and advanced maturity (Chapter 5).

Whether muscular hypertrophy occurs in mature animals (with an accompanying increase in total muscle weight) is still open to debate. However, in light of the results of these experiments, hypertrophy may be due only to changes in muscle distribution. The apparent susceptibility of human muscles to hypertrophy may be an exception. Two studies on male and female body-builders indicated that muscular hypertrophy only occurred in muscles of the upper body (Katch *et al.*, 1981; McDonald and Carlson, 1987). This may illustrate growth towards a mature muscle distribution; under normal conditions, upper body muscles are not used extensively by most modern-day humans, and perhaps remain at an immature distribution without physical stimulus. In any case, muscular hypertrophy represents little value to animal production since there are no gains in total muscle weight, and the small shifts in muscle distribution are not sufficient to compensate the producer for increased feed costs.

Total bone weight and bone weight distribution

The effects of exercise on bone distribution were more pronounced in the load-carrying and progressive-resistance-exercised sheep (Chapters II, III and IV) than in the endurance-exercised sheep (Chapter V). This may be attributed to the type of exercise since compressional forces are thought to have more effect than tensile forces on bone growth (Kiiskinen and Heikkinen, 1978). However, the load-carrying sheep and progressive-resistance-exercised sheep started the experiments at a much lower live weight (23-33 kg) than the endurance-exercised sheep (67kg). Bone weight was probably closer to its mature distribution at the heavier live weight and less susceptible to the effects of exercise (Kiiskinen, 1977).

The effects of exercise on bone distribution also appeared to be two-fold; both a direct effect of exercise and an acceleration of growth towards a mature distribution. In Chapter II, load-carrying sheep had a larger proportion of bone weight in the appendicular skeleton, probably as a direct response to the load carried. Conversely, when load-carrying sheep were allowed to feed *ad libitum*, a larger proportion of bone was found in the axial

skeleton, similar to differences which occur as a result of a later stage of maturity (Butterfield and Thompson, 1983). A later stage of maturity was indicated by the reduced proportion of cervical vertebrae evident in endurance-exercised sheep.

Load-carrying was found to reduce the length and increase the width of the metacarpal bone (Chapter III). The effects of the various types of exercise and initial live weights on bone shape were not investigated. However, the load-carrying experiments in young sheep would probably have had more influence on bone shape than the endurance-exercise in older sheep (Tulloh and Romberg, 1963; Kiiskinen, 1977; Kiiskinen and Heikkinen, 1978; Martin *et al.*, 1981).

Total fat weight and fat weight distribution

In most of the experiments, exercise had no effect on total fat weight. However, load-carrying sheep had a lower total fat weight (Chapter III) which was associated with a lower carcass weight (Berg and Butterfield, 1976; Butterfield *et al.*, 1984). The distribution of fat among the three fat depots (SCF, IMF, PRCF) was not significantly altered as a result of exercise. Fat distribution does not differ between sheep at different stages of maturity (Butterfield and Thompson, 1983). However, a rapid accumulation of SCF occurred during the first 6 weeks of endurance exercise (Chapter V), perhaps due to an exercise-induced acceleration of growth.

These results lend little support to reduced carcass fat as a benefit of exercise in young, growing animals. However, analysis of the carcass (Chapter III) and of individual muscles (Chapter V) indicated a reduction in chemically determined fat (presumably intramuscular fat). Thus, the main effect of exercise on fat content in the carcass is a reduction of intramuscular fat in young growing animals.

Dissectible connective tissue

Surprisingly, exercise had a very large influence on the proportion of dissectible connective tissue, particularly the *ligamentum nuchae*. The *ligamentum nuchae* was more than

twice as heavy in the load-carrying sheep (Chapter 2), and was significantly lighter in the endurance-exercised sheep (Chapter 5). The *ligamentum nuchae*, composed mainly of elastin and collagen, supports the weight of the head; changes in head weight did not parallel the changes in weight of the *ligamentum nuchae*. The increase in *ligamentum nuchae* weight in load-carrying sheep was thought a functional response to the decrease in the proportion of neck muscles which support the head. Increased tendon and ligament weights have been found previously (Tipton *et al.*, 1975; Kiiskinen, 1977), although they were thought to result from an exercise-induced acceleration of growth (Kiiskinen, 1977). The reason for the lower *ligamentum nuchae* weight in endurance-exercised sheep is unknown. However, it may be related to an accelerated collagen metabolism (higher turnover rate of collagen in growing exercised animals; Souminen *et al.*, 1980).

C. CELLULAR CHANGES

Myofiber volume

Increases in muscle cell diameters have been used as a measurement of muscular hypertrophy (MacDougall *et al.*, 1979; Schantz *et al.*, 1981). However, the consistently shorter sarcomeres of muscles from endurance-exercised sheep indicated that muscle diameters must be interpreted with caution. Shorter sarcomeres may result in misleadingly large diameters; Goldspink (1964) reported larger fiber diameters in exercised mice even though there was no increase in muscle weight.

Post-mortem pH decline

Muscles from endurance-exercised sheep generally had lower rates of post-mortem pH decline, indicating either slower post-mortem glycolysis or a change in the buffering capacity of the muscle. Three possible mechanisms were suggested: (1) a shift in glycolytic enzyme activities to favour aerobic metabolism; (2) decreased stress in the endurance-exercised sheep at slaughter; (3) an increase in the buffering capacity (specifically carnosine) of the muscle.

Regardless of the mechanism, the lower rates of post-mortem pH decline were thought responsible for the shorter sarcomeres in endurance-exercised sheep.

Fiber type composition

At the cellular level, changes associated with exercise could also be explained by an acceleration of growth. Endurance exercise resulted in an increase in proportion of slow oxidative fibers. These changes paralleled changes normally seen in fiber type composition as an animal ages.

D. MEAT QUALITY

Tenderness

Exercise increased meat tenderness (Chapters IV and V). The increase in tenderness has been previously associated with a dilution of collagen due to an increase in myofibrillar protein content (Mitchell and Hamilton, 1933). Although there was an increase in myofibrillar protein content, there was also a decrease in collagen content. Since there is evidence that exercise can both increase myofibrillar protein content (Mitchell and Hamilton, 1933; Helander, 1961) and decrease collagen content (Bailey, 1972; Kruggel and Field, 1974) it is difficult to identify the exact nature of the changes in these experiments.

E. IMPLICATIONS TO ANIMAL SCIENCE AND EXERCISE PHYSIOLOGY

Improvements can be made to experiments in both animal science and exercise physiology. The response of an animal to exercise occurs at many levels and can vary with the type of exercise (high intensity vs. low intensity), the length of time on exercise, and the ability of the animal to perform the exercise. Animal scientists have generally paid little attention to these aspects of exercise; experiments which concluded exercise had no effects on meat animals (Murray *et al.*, 1974a; Butterfield, cited in Berg and Butterfield, 1976) probably lacked sufficient intensity to produce changes. In addition, the degree of maturity of the

animal (mature vs. growing), the growth patterns of tissues, and the basis of comparison (same live weight, same total muscle weight, etc.) can influence the apparent response to exercise. Careful consideration of all these factors should be included in any exercise study. The present studies suggest that exercise can accelerate growth in actively growing tissues; at a given live weight, exercised animals may even appear to have a composition similar to a later stage of maturity.

In meat production, low intensity exercise may be useful. Although exercised animals consume slightly more feed to maintain similar growth rates, the increase in meat tenderness achieved through exercise could be beneficial. Unfortunately, the benefits of increased tenderness are only recognized at the consumer level; meat producers would be rewarded by increased feed costs. The negative aspects of exercise on production (decreased growth rate, lower yields of lean meat) were only associated with high intensity exercise. Thus, for the time being, exercising meat animals is not commercially viable. However, with increased awareness of an animal's well-being, more pressure may be put on producers to provide exercise to confinement-reared animals. This thesis may help answer some of the related questions.

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APPENDIX 1 - HISTOLOGICAL METHODS

A. Sample Preparation

Muscles were removed from the right side of the sheep carcass as soon as possible after slaughter and were kept on ice until subsampling took place. Small samples of muscle (less than .5 cm in diameter and less than 1 cm in length) were cut parallel to the grain of the muscle. The location of the subsample was constant for each muscle within an experiment. The samples were then placed on cryostat chucks perpendicular to the grain of the muscle fibers. Samples were covered in O. C. T. embedding medium (Ames) and frozen quickly in isopentane cooled to a semi-solid state in liquid nitrogen. Samples were maintained at -20° C until sectioned in an Ames Cryostat. Ten micron sections were cut on the cryostat, picked up on glass slides and allowed to air dry for approximately 30 minutes prior to staining.

B. Myosin ATPase

Skeletal muscle fibers differ in their speed of contraction and capacity to split ATP (Vander et al., 1980). The premise of the myosin ATPase stain is that the speed at which a fiber contracts is directly related to the rate at which myosin splits ATP, which is proportional to the myosin ATPase activity (Swatland, 1984). The myosin ATPase activity is monitored histochemically. The relationship between contraction speed and histochemical myofibrillar ATPase activity has been confirmed by Zeman and Wood (1980). The following steps, with their chemical interpretation (Bancroft, 1977), were modifications presented by Guth and Samaha (1970) of the technique introduced by Padykula and Herman (1955).

1. Sections were fixed in a formalin solution to stabilize the proteins in their *in vivo* positions.
2. Sections were pre-incubated in a CaCl_2 buffer (pH 10.4). The Ca^{++} activated the fibers by binding to troponin, which causes a shift in the position of tropomyosin, thus exposing the cross-bridge binding sites on actin.

3. The 'activated' sections were incubated in a buffer (pH 10.4) containing ATP for approximately 50 minutes. During this period, the myosin ATPase enzyme released phosphate from the ATP substrate. The degree of phosphate production was dependent on the activity of the enzyme.
4. The incubated sections were washed to stop the reaction and placed in a CoCl_2 solution. The phosphate ions combined with the Co^{++} , producing a precipitate of cobalt phosphate.
5. The sections were then exposed to a dilute solution of ammonium sulphide. The cobalt phosphate reacted with the sulphide to form cobalt sulphide which appeared as a dark brown precipitate at the site of enzyme activity.
6. The stained sections were rinsed in distilled water, dehydrated in graded ethanol and cleared in xylene prior to mounting using dilute Permount.

At the alkaline pre-incubation pH, fast fibers have alkali-stable ATPase and slow fibers have alkali-labile ATPase. Hence the fast 'white' fibers stained darkly, and the slow 'red' fibers did not stain. The 'intermediate' fibers also stained darkly.

C. NADH Dehydrogenase

Muscle fibers differ in their ability to produce ATP, either through glycolysis or oxidative phosphorylation (Krebs cycle and the electron transport chain) (Vander et al., 1980). The enzymes of the electron transport chain are associated with the inner membrane of the mitochondria (Lehninger, 1970). Histological staining can be used to localize the activity of these enzymes and thus localize the mitochondria within a cell. The number and location of the mitochondria gives an indication of the oxidative capacity of a cell. Localization of the enzyme NADH dehydrogenase was accomplished as follows (Bancroft, 1977).

1. Sections were incubated at 37° C for 30 minutes in a Tris buffer solution (pH 7.4)

containing the co-enzyme NADH and nitro-blue tetrazolium (N. B. T.). During this incubation, the enzyme NADH dehydrogenase and the co-enzyme NADH participate in the transfer of H^+ ions to the tetrazolium salt. The reduction of the tetrazolium salt produces blue formazan granules which are deposited at the site of enzyme activity.

2. Sections were dehydrated in graded alcohols, cleared in xylene and mounted with Permount.

Novikoff et al. (1961) confirmed that N. B. T. staining indicates the intracellular localization of oxidative enzymes at the level of light microscopy. Some formazan deposition artifacts occur on lipid-aqueous interfaces.

D. Fiber Typing

Slides were examined under a Leitz-Wetzlar light microscope. Five to ten areas were randomly recorded per slide (not serial sections) on a Sony KCA-60K video cassette (camera lens 25X, objective lens 25X). For the myosin ATPase stain, fibers were subjectively categorized as either fast (dark staining) or slow (not-stained). For the NADH dehydrogenase stain, fibers were subjectively categorized as either oxidative (many mitochondria stained darkly), intermediate (mitochondria darkly stained, mainly around outside edge of cell), or glycolytic (relatively few mitochondria stained, scattered throughout cell).

APPENDIX II - DYSPROSIUM AS A DRY MATTER DIGESTIBILITY MARKER

The rare earth element dysprosium is a useful digestive marker due to its indigestibility and its ability to become tightly bound to plant material (Ellis, 1968). The use of dysprosium as a digestibility marker has been developed by Kennelly et al. (1980a) on the basis of grab samples from feed and fecal material.

A. Incorporation into the Feed

Fifty grams of dysprosium chloride ($\text{DyCl}_3 \cdot 6\text{H}_2\text{O}$) were ground and mixed with 1 kg of finely ground barley. This mixture was added to 5 kg of less finely ground barley and mixed. This was then mixed with 40 kg of coarsely ground barley and incorporated into 1600 kg of feed. The final concentration of dysprosium in the feed was 13.47 p.p.m..

B. Sampling Procedure

The sheep were started on the feed containing dysprosium two weeks prior to starting the pre-trial training period. During the pre-trial and trial periods, grab samples were attempted three times daily, on two consecutive days of the week (Saturday and Sunday). The feces collected on these six collections were pooled and treated as a single sample per sheep. A total of 10 samples were collected per sheep; two during the pre-trial period and eight during the trial. The samples were stored at -20°C until analysed.

C. Sample Preparation and Analysis

The samples were freeze dried and ground to a fine powder using a Janke and Kunkel analytical mill. Duplicate samples of the ground feces and feed were dried for 48 hours at 110°C to obtain dry matters. Samples of feed (1-2 grams) and feces (≤ 1 gram) were weighed accurately into plastic irradiation vials. These vials were packed with table sugar (sucrose) and heat sealed. The feed and fecal samples were analysed for dysprosium by I.N.A.A. (Instrumental Neutron Activation Analysis) at the University of Alberta

SLOWPOKE facility (Kennelly et al., 1980b). The ratio of dysprosium in the feed to dysprosium in the feces on a dry matter basis resulted in estimates of dry matter digestibility.

APPENDIX III - DRIP LOSSES, MECHANICAL TENDERNESS AND ULTIMATE PH

A. Sampling Procedure

After 48 hours of storage at 6° C the *semimembranosus* (SM; Chapter V) and the *vastus lateralis* (VL; Chapter IV and V) muscles were removed from the left side of the carcass. The muscles were weighed and linear measurements of length, width, depth and circumference were recorded. The distal and proximal ends of the muscles were marked. The muscles were then double-wrapped in freezer paper and stored in labelled plastic bags at -20° C until analysed.

B. Drip Losses

After 9 to 13 weeks storage in the freezer, the SM and VL were thawed in a 3° C cooler (40 and 24 hours, respectively). Thaw drip losses were recorded and a portion of the distal end of the muscles was removed for the determination of ultimate pH. Thermocouples were placed in the geometric center of the muscles and the muscles were then cooked in a 177° C oven to an internal temperature of 70° C. Muscles were cooled to an internal temperature of 50° C before drip and volatile cooking losses were recorded. The muscles were stored overnight in a 3° C cooler.

C. Warner Bratzler Shear and Kramer Press - Ground

The following day the external surfaces of the cooked muscles were trimmed. The SM muscles were sliced on an automatic meat slicer into three 1.27 cm slices parallel to the grain of the muscle fibers. The anatomically deepest slice was cut into core samples 2.54 cm x 1.27 cm x 1.27 cm. A similar procedure was followed for the VL muscles except all the slices were used for core samples. The core samples were sheared on an Instron 4201 equipped with a Warner Bratzler Shear cell. Sheared core samples plus the remaining slices were ground and three 20 g subsamples were put through the Ottawa Texture Measuring System (O.T.M.S.) equipped with a Kramer Shear cell.

D. Ultimate pH

Twenty grams of muscle from the SM and 10 g of muscle from the VL were added to deionized, distilled water (100 and 50 ml, respectively). Samples were homogenized in a blender for 60 seconds and then were filtered through Whatman No. 1 fluted filter paper. The pH was recorded on duplicate samples of the filtrate using a Fisher Accumet 610A pH meter.

APPENDIX IV - CHEMICAL ANALYSIS

A. Proximate Analysis

Samples were prepared for proximate analysis in the following manner. Muscle samples were weighed into tared 600 ml beakers. Twenty ml of water was added, the beakers were covered with foil and the samples were autoclaved for 14 hours at 110° C and 15 psi. After cooling, the contents of the beakers were reduced to a homogenous puree using a Polytron PCU-2 homogenizer. The samples were then transferred to pre-weighed aluminum trays, washed with water to remove all residue, frozen (-20° C) and freeze-dried for five days. The freeze-dried weight was recorded prior to grinding the sample in a Janke and Kunkel analytical mill. Samples were taken from the powdered muscle for determination of dry matter, nitrogen, fat and ash according to AOAC (1980).

B. Protein Fractionation

Helander's procedure (1957) is capable of breaking muscle tissue into myofibrillar, sarcoplasmic, stromal and non-protein nitrogen fractions. However, the extraction of myofibrillar protein becomes incomplete after 48 hours storage, therefore, the procedure of Perry and Corsi (1958) was used. This procedure yields only the myofibrillar fraction; the other fractions are extracted leaving behind the myofibrillar protein.

Minced muscle samples (approximately 0.1 g) were weighed into test-tubes. Samples were homogenized in 5 ml of a borate-KCl-EDTA buffer (pH 7.1). One milliliter of homogenate was reserved for total protein determination. The remaining homogenate was centrifuged at 800 xG for 15 minutes and the supernatant was discarded. The protein pellet was resuspended in 5 ml of borate-KCl-EDTA buffer and centrifuged at 800 xG for 15 minutes. The supernatant was discarded and the protein pellet was resuspended in 5 ml of borate-KCl buffer (no EDTA) and centrifuged at 800 xG for 15 minutes. The protein pellet was resuspended in 1 ml of borate-KCl buffer.

Protein concentrations were determined using the method of Lowry et al. (1951), adapted for use on the Brinkmann PC800 Colorimeter. An aliquot of the homogenate (0.4 ml) was brought to a final volume of 2 ml with 1N NaOH and heated in a water bath for 2 hours at 37° C to hydrolyse the protein. After heating, the samples were vortexed and inspected to ensure there were no solid particles remaining. Four hundred μ l of the sample was brought to a final volume of 8 ml with distilled water. Triplicate samples of standards (12.5, 25, 37.5, 50 μ g/ml) were prepared using a solution of bovine serum albumin (BSA; 100 μ g/ml). Appropriate aliquots of the homogenate (selected to fall within the range of the standard curve) were analysed in triplicate to determine their protein content.

All tubes were brought to a final volume of 2 ml with distilled water. A solution containing copper sulphate and sodium tartrate (3 ml) was added to the tubes at timed intervals, followed by vortexing. After exactly 15 minutes, 0.4 ml of Folin reagent was added while mixing on the vortex. Colour was allowed to develop for exactly 30 minutes before reading the absorbance at 750 nm. At alkali pH, copper reacts with protein. When the Folin reagent is added, the copper treated protein reduces the Folin reagent, resulting in the development of a dark blue colour.

C. Total and Soluble Collagen

Sample preparation combined the procedures of Stegemann and Stalder (1967) and Culler et al. (1978). For total collagen, 4 g of scissor-minced muscle were placed in 100 ml glass pyrex tubes. For soluble/insoluble collagen, 4 g of scissor-minced muscle were weighed into centrifuge tubes and 20 ml of buffer (0.1M NaPO₄, pH 7.0>) was added. The soluble/insoluble collagen samples were then heated in a 72-74° C water bath with occasional stirring until the sample temperature reached 70° C. The samples were cooled in an ice bath for 10 minutes followed by centrifugation at 10,000 xG for 15 minutes. The supernatant (10 ml; soluble collagen) and the residue (insoluble collagen) were separated and individually transferred to 100 ml glass pyrex tubes. Forty ml of 6N HCl was added to all of the glass pyrex tubes and the samples were autoclaved (12 hours for total and insoluble collagen

samples, 6 hours for soluble collagen samples) at 19 psi and 125° C. The samples were cooled, decolorized with 2-3 g of a 1:2 (w:w) mixture of activated charcoal and dry Dowex AG 1-X8 ion exchange resin and then filtered.

After filtration, an aliquot of filtrate (5 ml for total and insoluble collagen and 10 ml for soluble collagen) was placed in 250 ml round bottom flasks. The HCl was evaporated using a Buchi Rotavapor RE120. The total collagen and insoluble collagen were resuspended in 20 ml of dilute acetate-citrate buffer (50 ml stock buffer in 450 ml of distilled water). Soluble collagen samples were resuspended in 10 ml of dilute acetate-citrate buffer. The stock buffer (pH 6.0) was composed of 45.7 g citric acid (anhydrous), 11.5 ml acetic acid, 120 g sodium acetate•3H₂O and 34 g sodium hydroxide dissolved in distilled water and brought to a final volume of 1000 ml.

A 5 ml aliquot of each sample was applied to a column containing approximately 3 ml of Dowex AG 50W-X8(H⁺) 200-400 mesh equilibrated with the dilute buffer (Stegeman and Stalder, 1967). Columns were washed twice with 2 ml of dilute buffer and samples were brought to a final volume of 10 ml.

Samples were analysed for hydroxyproline using the procedure of Stegemann and Stalder (1967). Triplicate samples (0.05, 0.10, 0.20, 0.30, 0.40 ml) of a standard solution (10 µg hydroxyproline/ml) were used to determine a standard curve. Triplicate samples of 150 µl for total collagen, 200 µl for insoluble collagen and 400 µl for soluble collagen were used for the hydroxyproline assay. All samples were brought to a final volume of 2 ml with a solution of 10% stock buffer and 20% propanol in distilled water. One milliliter of a Chloramine-T solution was added to each tube to oxidize the hydroxyproline. After 20 minutes 1 ml of an aldehyde/perchloric acid solution was added, the mixture was thoroughly shaken and placed in a water bath (60° C) for 15 minutes. The *p*-dimethylaminobenzaldehyde suspended in the aldehyde/perchloric acid solution reacts with the oxidized form of hydroxyproline to produce a yellowish-orange chromophore. After cooling to room temperature, the absorbance at 550 nm was read on a Brinkmann PC800 Colorimeter.

Hydroxyproline values were converted to total and insoluble collagen using a factor of 7.25 (Goll et al., 1963) and to soluble collagen using a factor of 7.52 (Cross et al., 1973). These values are based on the assumptions that some of the hydroxyproline in the total and insoluble collagen fractions came from elastin (collagen to elastin ratio, 3:1; hydroxyproline in collagen 13.3%; hydroxyproline in elastin 1.5%) and that no elastin was present in the soluble collagen fraction.

D. Myofibrillar Fragmentation Index (MFI)

During post-mortem storage myofibrillar fragmentation occurs at or near the Z-disk (Olson et al., 1976). Davey and Gilbert (1969) developed the myofibrillar fragmentation index based on the evidence that the degree of myofibrillar fragmentation was related to the light absorbance of a myofibrillar suspension. Culler et al. (1978) reported that the MFI was a more important predictor of tenderness than either collagen solubility or sarcomere length.

MFI's were determined using the Culler et al. (1978) modification of Davey and Gilbert (1969). Three cores (1.27 cm diameter) were removed from the frozen muscle. The samples were scissor-minced and visible fat and connective tissue was removed. Four grams of minced muscle were weighed into a flask and 40 ml of isolating medium (100 mM KCl, 20 mM K_3PO_4 , 1 mM EDTA, 1 mM $MgCl_2$ and 1 mM NaN_3) chilled to 2° C was added. The sample was homogenized for 30 seconds at 20,000 rpm in a Virtis '45' Tissue Homogenizer. The homogenate was centrifuged at 1000 xG for 15 minutes and the supernatant was discarded. The residue was resuspended in 40 ml of isolating medium, centrifuged at 1000 xG for 15 minutes and the supernatant was discarded. The protein pellet was resuspended in 10 ml of isolating medium and passed through non-standard 16 and 20 nylon mesh strainers having a respective pore size of 1.69 mm^2 and 0.46 mm^2 . The residue was washed with 10 ml of isolating medium resulting in a 20 ml myofibrillar protein suspension. Protein concentrations of the solutions were determined using the method of Lowry et al. (1951) which was described previously. The solutions were then diluted with isolating medium to a protein concentration of $0.50 \pm 0.05\text{ mg/ml}$. The diluted myofibrillar suspension was

vortexed, poured into a cuvette and the absorbance was measured at 540nm with a Bausch and Lomb Spectronic 20 colorimeter. Absorbance was multiplied by 200 to give a MFI value.

E. Glycogen Assay

The residual post-mortem glycogen content was determined using the procedure described by Lo et al. (1970). Five samples weighing approximately 35-50 μg were removed from the gastrocnemius muscle 48 hours after slaughter. The weighed samples were placed in capped test-tubes and quick frozen in a mixture of dry ice and methanol. The samples were stored at -20°C until assayed.

Test-tubes were kept on ice after removal from the freezer. A small aliquot (0.5 ml) of 30% KOH saturated with Na_2SO_4 was added to the tubes. The capped test-tubes were heated in a boiling water bath for 20-30 minutes until a homogenous solution was obtained. After cooling on ice, 0.6 ml of 95% ethanol was added to precipitate the glycogen. The test-tubes stood on ice for 30 minutes and then were centrifuged at 840 xG for 30 minutes. The supernatants were carefully drawn off with a pasteur pippette and the remaining glycogen precipitate was dissolved in 3 ml of distilled water.

Standards (25, 50, 75, 100 μg glycogen/ml) were prepared in triplicate from a stock solution of 100 μg glycogen/ml. Appropriate aliquots of the glycogen samples, falling within the range of the standard curve, were assayed in triplicate. All samples were brought to a final volume of 1 ml. One milliliter of 5% phenol was added and the tubes were vortexed. Immediately after, 5 ml of concentrated sulphuric acid were added to the tubes by directing the stream against the liquid surface to ensure good mixing. Tubes were allowed to cool for 10 minutes before gently mixing (tubes were tipped back and forth between thumb and forefinger). Tubes stood for 20 minutes in a water bath at 25°C before reading their absorbance at 490 nm on a Brinkmann PC800 Colorimeter. The concentrated sulphuric acid hydrolysed the glycogen; the resulting glucose reacted with two molecules of phenol to produce a yellowish-orange colour.

APPENDIX V - OXYGEN ANALYSING SYSTEM

A. Equipment

A Beckman OM11 oxygen analyser was used in a flow-through system. This machine samples on a wet air basis (air is not dried in drierite before sampling). The Fischer and Porter flowmeter (Series 10A 3000) had a rated maximum capacity of 765 litres of O_2 per minute. The flow meter was set at 25% of its maximum capacity. A Fischer Recordall (Series 5000) recorded the O_2 decrement with a full scale deflection equal to a 5% O_2 decrement. Wet and dry bulb temperatures were recorded using a Bailey Instruments thermocouple temperature indicator. A mixing chamber (a large enclosed plastic container) was placed in the line to dampen background deflections in O_2 decrements due to the animals breathing. A Gast rotary vane pump (Model 1022-V103-G272X) was used to draw air through the system. A home-made mask, constructed of plastic, was fitted to the sheep's muzzle with neoprene and strapped on with a nylon strap which buckled behind the sheep's ears.

B. Measurements

The equipment was allowed approximately one half hour warm up time while a baseline atmospheric oxygen content was established at 20.95% O_2 . Resting oxygen consumptions were established over a 20 minute period while the sheep were standing on the treadmill. Sheep were run on the treadmill at a speed which elicited an oxygen decrement equivalent to 75% of their previously determined VO_2 max. Average oxygen decrements over the resting period and time running on the treadmill were used to calculate the energy output. Wet and dry bulb temperatures were recorded for each animal while resting and running. Daily barometric pressure was also recorded.

C. System Calibration

A nitrogen injection was performed to calibrate the system (Young et al., 1984). The mean nitrogen recovery was 0.8000, therefore a correction factor of 1.2 was applied to all the numbers.

D. Calculations

The steady state oxygen decrements were converted to litres of oxygen consumed per kg live weight per minute at standard temperature and pressure. Caloric equivalents of oxygen (MacLean, 1972) were used to convert oxygen consumptions to heat production per kg of metabolic body weight ($BW^{.75}$) per minute. Heat production values were corrected for system losses by multiplying by 1.2.

APPENDIX VI - POST-MORTEM PH AND TEMPERATURE DECLINE

The pH and temperature decline were monitored using a Philips combination pH electrode for puncture measurements on a Corning pH/temperature Meter 4.

A. Calibration

Calibration of the electrode was done at the beginning of each slaughter and was checked between animals. A drift of ± 0.05 pH units was allowed between calibrations. The Corning pH/temperature Meter 4 was standardized at two temperatures; room temperature (20°C) for the first readings after slaughter and cooler temperature (6°C) for all other readings. The temperature of the pH 7.00 standard was recorded and the automatic temperature adjustment dial was set to the recorded temperature. The pH electrode was then placed in the pH 7.00 standard and allowed one minute to equilibrate. If necessary, the meter was calibrated to read pH 7.00. The same steps were repeated using the pH 5.00 standard. The pH 7.00 standard was re-checked. A drift of ± 0.05 pH units at pH 5.00 was tolerated if the pH 7.00 standard was accurate to ± 0.01 .

B. PH and Temperature Readings

The time of exanguination was recorded and the first readings were taken as soon as the sheep had been eviscerated and skinned (T_0). Muscles were always tested in the following order: *semimembranosus* (SM), *vastus lateralis* (VL), *semitendinosus* (ST), *psoas major* (PM) and *gastrocnemius* (G). In order to reach the PM, much of the fat covering this muscle had to be removed. It was also necessary to reflect the *biceps femoris* to gain access to the G. A small incision was made (1-2 cm) in the muscle with a scalpel. The pH and temperature electrodes were inserted approximately 2 cm into the incision, making sure that they did not touch. The pH meter was then calibrated for the temperature of the muscle and the temperature and pH were recorded. The pH and temperature electrodes were rinsed with

distilled water before use in another animal. Temperature and pH readings were recorded at T_0 and 1, 3, 6, 9, 12 and 24 hours after T_0 .

APPENDIX VII - MICROSCOPE TECHNIQUES

A. Sample Preparation

Forty-eight hours after slaughter, two thick sections (4 mm x 1 cm x 2 cm) of each muscle (SM, VL, ST, PM, G) were fixed in 10% formalin (4% formaldehyde) and were stored in a fumehood until analysed. The samples were suspended in 50 ml of 10% glycerol brought to volume with methanol. The samples were macerated in a Virtis '45' Tissue Homogenizer at 20,000 rpm for 30-60 seconds. A sample of the myofiber suspension was removed for determination of muscle fiber diameters. Maceration of the myofiber suspension was continued until myofibrils dominated the suspension (approximately 1 minute). A sample of the myofibril suspension was then removed for determination of sarcomere lengths. This preparation technique was similar to the technique described by Swatland (1980).

B. Muscle Fiber Diameters

Two slides were prepared of each myofiber suspension by placing a drop of the suspension on a slide and covering the drop with a coverslip. The samples were examined under a Leitz-Wetzlar light microscope (ocular lens 12.5X, objective lens 25X). Diameters were measured using an ocular grid which had markings every $3.57\mu\text{m}$. Thirty diameters were measured on each slide for a total of 60 diameters measured per muscle.

C. Sarcomere Lengths

A drop of myofibril suspension was placed on a well slide and covered with a coverslip. A drop of immersion oil was placed on the coverslip and the slide was examined under the same Leitz Wetzlar microscope set up for phase contrast work (ocular lens 12.5X, objective lens 100X). Isolated myofibrils were recorded on a Sony KCA-60K video cassette (camera lens 25X, objective lens 100X). A micrometer grid was recorded for conversion of measured sarcomere lengths to actual lengths. An electronically produced grid (distortion free) was also recorded to check the degree of monitor distortion. An editing monitor

(Department of Radio and Television, University of Alberta) was used to play back the recorded tapes. Very little distortion occurred using this monitor except around the extreme edges. The edges were avoided when measuring sarcomere lengths. Sarcomere lengths were measured using modified calipers, and were converted to real lengths using a conversion factor of $1.277 \mu\text{m}/\text{cm}$ measured.

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